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Philadelphia College of Osteopathic Medicine
School of Professional and Applied Psychology
Department of Clinical Psychology

THE RELATIONSHIP BETWEEN INSOMNIA, SLEEP CONTINUITY
DISTURBANCE, SLEEP-RELATED DAYTIME DYSFUNCTION, PROBLEM
ENDORSEMENT, AND AGING IN A COMMUNITY-BASED SAMPLE

By Julia T. Boyle

Submitted in Partial Fulfillment of the Requirements for the Degree of

Doctor of Psychology

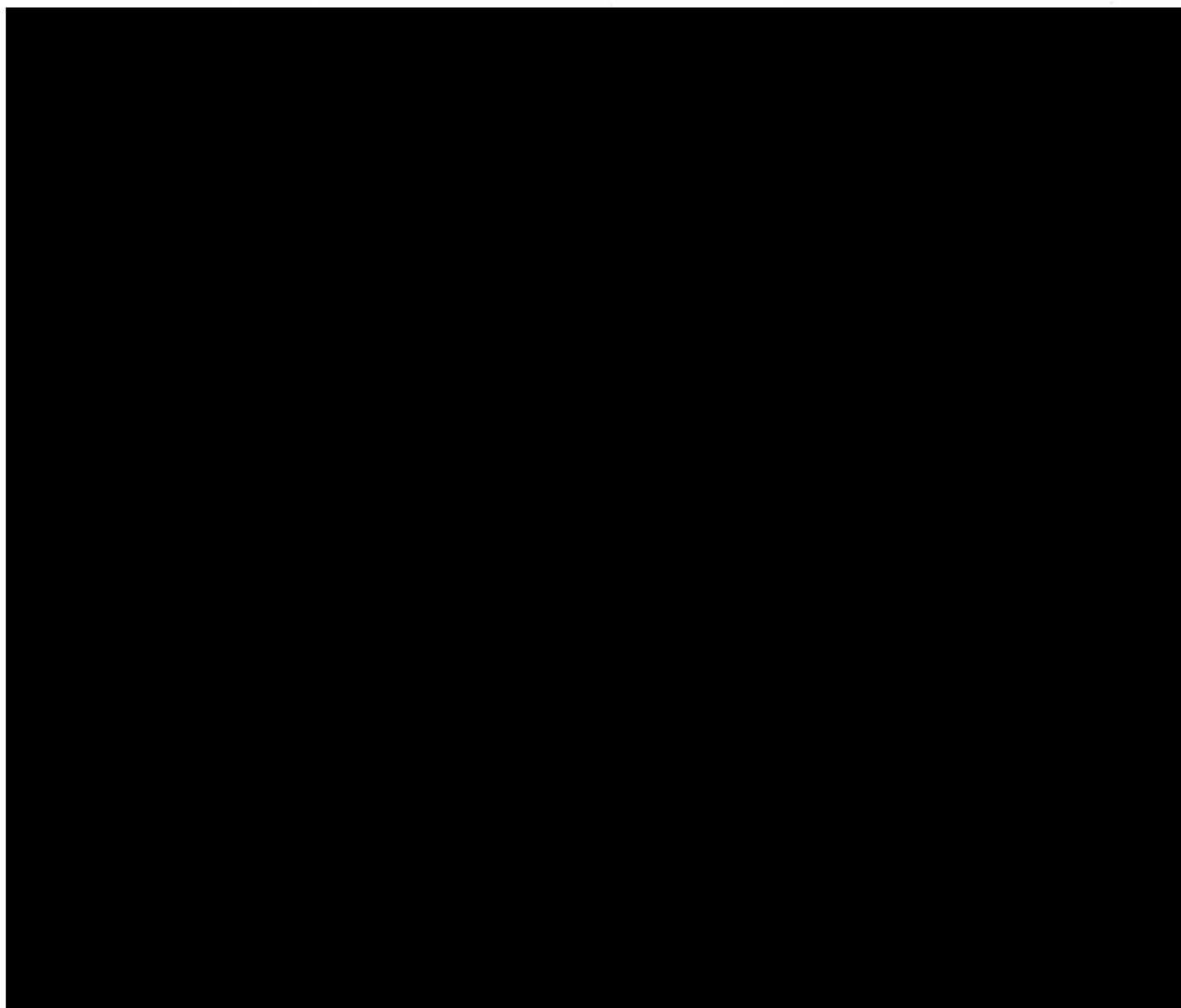
June 2020



PCOM SCHOOL OF PROFESSIONAL AND APPLIED PSYCHOLOGY™



DISSERTATION APPROVAL



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ABSTRACT

Although older adults experience greater difficulty initiating sleep, maintaining sleep, or waking up earlier than intended, they are less likely to report that they perceive this to be a problem. The discordance between sleep disorder symptoms and the reported perception that they are troubling is unusually prevalent among older adults. This may be problematic, in that individuals who do not perceive their sleep continuity disturbances (SCDs; difficulty falling asleep, staying asleep, or waking up earlier than intended) to be problems are less likely to seek treatment. Untreated SCD has been shown to be a risk factor for the development or exacerbation of multiple medical and psychiatric disorders such as cardiovascular disease, stroke, dementia, and depression. This study aimed to identify the relationship between age, SCD, sleep-related daytime dysfunction, and the perception that these symptoms are problematic. The study utilized a cross-sectional group comparison approach to assess for age differences in relation to SCD, sleep-related daytime dysfunction, and percent problem endorsement in an archival, community dataset comprised of individuals between 18 and 89 years of age, with sleep complaints. The results indicated that, as expected, SCD worsens with age (except sleep latency); sleep-related daytime dysfunction did not worsen with age except for concentration; and, contrary to expectation, problem endorsement increased with age (except for sleep latency). Findings illustrate the importance of comprehensive sleep assessments, preferably with the inclusion of the question: Is this a problem?

CHAPTER 1: INTRODUCTION

Statement of the Problem

Sleep disorders affect up to 70 million adults in the U.S., although it is likely that many of these individuals never sought treatment or received diagnoses (Colten & Altevogt, 2006). Ultimately, the high prevalence of sleep disorders lends itself to a significant and unnecessary economic burden worldwide (Colten & Altevogt, 2006; Ozminkowski, Wang, & Walsh, 2007). For example, Hillman, S. Mitchell, Streatfield, Burns, Bruck, and Pezzullo (2018) assessed the financial and non-financial costs of inadequate sleep in Australia between 2016 and 2017. The authors defined financial costs as health care, health-related care that was obtained outside the health care system, productivity losses, work and vehicle accidents not related to medical conditions, and additional losses related to lost tax revenue and welfare payments. The non-financial costs encompassed a “loss of well-being” or reduction in quality of life due to changes in cognitive function, psychomotor skills, mood, physical health, and emotional health due to inadequate sleep. The authors further defined inadequate sleep as “difficulties with sleep initiation, maintenance, or quality associated with the presence of impaired daytime alertness on several days a week or more” (p. 2). The researchers determined that insufficient sleep could be due to sleep disorders, specifically obstructive sleep apnea (OSA), insomnia disorder, and restless leg syndrome (RLS; Hillman et al., 2018).

The U.S. has the highest economic burden in the world related to insufficient sleep, falling between \$280 and \$411 billion per year. Although this is largely due to the size of the U.S. economy, researchers predict that this cost will worsen to between \$299 and \$433 billion annually by 2020 and between \$318 and \$456 billion by 2030 (Hafner,

Stepanek, J. Taylor, Troxel, & van Stolk, 2017). Added to these daunting statistics is the fact that insufficient sleep and sleep disorders in general do not only impact the U.S. Total economic costs of inadequate sleep in Australia between 2016 and 2017 was \$42.21 billion, of which \$17.88 billion was related to financial costs and \$27.33 billion related to non-financial costs. Of the financial costs, \$160 million was due to direct health costs of sleep disorders and \$1.08 billion was related to treatment of medical conditions worsened by inadequate sleep (Hillman et al., 2018). This further indicates that insufficient sleep and sleep disorders are a global concern that must be addressed.

Costs related to sleep disorders directly increase economic burden. The direct costs of evaluating and treating sleep disorders for individuals in the U.S. suffering from OSA alone is between \$17.5 billion and \$3 billion (Sassani et al., 2004). The indirect costs include medical, psychological, and neurological comorbidities, all of which have been proven related to poor sleep (Grandner, 2012; Ozminkowski et al., 2007). This leads to added health care costs within the workplace where individuals who “always” experience sleep disturbances and sleep-related daytime dysfunction can cost employers \$5206 per person per year. Alternatively, individuals who seldom experience sleep disturbances and associated sleep-related daytime dysfunction cost employers \$551 per person per year (Hui & Grandner, 2015). Even more concerning is that older individuals are less likely than younger individuals to report sleep continuity disturbances (SCDs) to their health care providers, thus incurring additional lifetime costs of \$3,831 for each patient under the age of 65 with untreated insomnia and \$4,647 for each patient over the age of 65 with untreated insomnia (Grandner, Martin, et al., 2012; Ozminkowski et al.,

2007). Without proper treatment, patients' sleep may worsen, which becomes increasingly problematic as humans age (Grandner, 2012).

Although some individuals may experience little to no changes in their sleep as they age, many others may experience significant changes. Specifically, as individuals age, they may experience greater difficulty falling and staying asleep, and encounter issues of waking up earlier than intended, defined as SCDs (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). The term SCD will be used in place of insomnia as it connotes difficulty falling asleep, staying asleep, and waking too early without necessarily meeting criteria for an insomnia disorder diagnosis, in that insomnia disorder requires the addition of significant dissatisfaction with total sleep time (TST) or sleep quality that is directly related to difficulty falling asleep, staying asleep or waking too early. Furthermore, an individual with insomnia disorder must exhibit clinically significant distress as a result of these sleep disturbances (American Psychiatric Association [APA], 2013). Finally, an insomnia disorder diagnosis requires that sleep disturbances must be present for at least 3 nights per week for at least 3 months. By using the term SCD instead of insomnia disorder, the present study will encompass the duration and frequency of all sleep disturbances with and without associated clinically significant distress (APA, 2013; Perlis, personal communication, May 2018).

Although older individuals are less likely than younger cohorts to clearly report these disturbances to their health care providers, these disturbances are highly prevalent amongst this population (Grandner, Martin, et al., 2012). Many recent findings may explain the discrepancy between sleep disturbances and sleep-related daytime dysfunction. For example, many older individuals are no longer working, so it is likely

that decline in TST may not impact their daily functioning as much as it would younger, working adults with the same symptoms. Furthermore, older individuals may consider these sleep disturbances to be a part of “normal” aging (Mander, Winer, & Walker, 2017). This inability to recognize sleep disturbance as a perceived problem could be detrimental for older individuals’ overall mental and physical health, as they may still suffer from impaired daytime functioning and a lowered threshold for medical and psychiatric illnesses such as cardiovascular disease, diabetes, dementia, obesity, stroke, anxiety, and depression as a results of SCD (Grandner, 2012).

As such, empirically supported research is needed to bring greater awareness of these risks to the aging population and their health care providers. Such knowledge could encourage health care providers to inquire about their older patients’ sleep on both an objective level (e.g., How many hours of sleep do you get each night, on average?) and a subjective level (e.g., Do you feel rested upon waking for the day?). Currently, as practiced, sleep assessment is not a consistent or comprehensive measure of patients’ health, despite its relevance to psychiatric, neurological, cardiovascular, and hormonal function (Grandner & Malhotra, 2015).

Much of the sleep literature has supported a strong relationship between sleep disturbance and aging, psychological, neurological, and medical comorbidities. This literature mostly evaluates these relationships between individuals with normal sleep and individuals with insomnia (Lichstein, Durrence, Riedel, D. J. Taylor, & Bush, 2013). Moreover, the existing sleep literature appears to assess either the severity of the SD (objective sleep measures) or sleep-related daytime dysfunction (daytime consequences of poor sleep; Morin, 1993; M. L. Perlis, personal communication, June 2018, February

2019). It is rare that researchers assess both SCD severity and symptoms of sleep-related daytime dysfunction (M. L. Perlis, personal communication, June 2018). Unfortunately, not all authors explicitly state these categories and others incorrectly assume that the presentation of an SCD must mean that it is perceived to be a problem for the participant. What has not yet been determined is the point at which aging individuals endorse SCD or sleep-related daytime dysfunction to be a perceived problem. Unfortunately, there is no current literature on this, despite the growing field of sleep and aging research.

The perception of SCD severity or sleep-related daytime dysfunction as a problem is additionally impacted by the misperception of what is considered “normal” aging (Mander et al., 2017). As individuals age, they may be less likely to clearly report these sleep disturbances or symptoms of sleep-related daytime dysfunction to their health care providers (Grandner, Martin, et al., 2012). To respond to these concerns, the present study sought to determine the relationship between age and the point at which individuals report perceiving SCD and sleep-related daytime dysfunction to be a problem.

Purpose of the Study

There is substantial evidence indicating clear relationships between certain medical and psychological disorders, aging, and sleep disturbance. Many sleep studies are limited, in that they focus on restricted age ranges, and few studies examine sleep across the life cycle. Furthermore, the existing data generally fall into two categories: SCDs, which focuses on sleep latency (SL), nighttime awakening (NWAK), wake after sleep onset (WASO), early morning awakenings (EMA), and total sleep time (TST); or sleep-related daytime dysfunction, which focuses on the adverse outcomes of poor sleep. To date, few studies have assessed the quantitative and qualitative aspects of insomnia

concurrently. No studies have inquired explicitly as to whether reported SCD or sleep-related daytime dysfunction are, concurrently, perceived to be problems.

Accordingly, the proposed study sought to identify the age-related differences between SCD, sleep-related daytime dysfunction, and problem endorsement in a large community sample that consisted of individuals between the ages of 18 and 89. The results of this study may be used to inform clinicians how to accurately assess and treat older adults. Specifically, this data may suggest that treatment recommendations should be made on the basis of symptom duration and frequency even in the absence of SCD, problem endorsement, or sleep-related daytime dysfunction.

Hypotheses

Hypothesis I. It was hypothesized that SCD would worsen with age and that this would be true especially for middle and late SCD (NWAK, WASO, and EMA). SCDs were operationalized as the duration and frequency of SCDs for initial, middle and late insomnia. SCDs were assessed through self-reported measures of SL, NWAK, WASO, EMA, and overall TST. The questions regarding SCDs can be found on items 33, 37, 41, 46, and 50 within Section 2 of the www.sleeplessinphilly.com survey (see Appendix B).

Hypothesis II. It was hypothesized that there would be a significant age group difference for symptoms of sleep-related daytime dysfunction, in that the older age groups (middle age and older adults) would report less symptoms of sleep-related daytime dysfunction. Sleep-related daytime dysfunction was operationalized as symptoms that directly pertain to daytime function within the source data (attention or concentration, restedness, general daytime function, fatigue, and sleepiness). Symptoms of sleep-related daytime dysfunction were evaluated individually and as a composite

variable created by the researchers. The questions about sleep-related daytime dysfunction were presented to the participants as five separate yes/no questions of daytime consequences, which can be found on items 55, 58, 59, 60, and 61 of Section 2 of the www.sleeplessinphilly.com survey (see Appendix B).

Hypothesis III. It was hypothesized that there would be a significant difference between age groups regarding participants' endorsement of sleep disturbances as perceived problems. Specifically, older age groups (middle age and older adult) would be less likely to report their SCD symptoms as problematic. Problem endorsement was operationalized by whether the participant viewed their SCDs as problems. Questions regarding problem endorsement can be found on items 35, 39, 43, 48, and 52 of the www.sleeplessinphilly.com survey (see Appendix B). Of note, these items followed questions related to SCD, so that they specifically ask participants whether they consider their SL, NWAK, WASO, EMA, and TST to be problems.

CHAPTER 2: REVIEW OF THE LITERATURE

What is Sleep?

There are many definitions of sleep. For example, in 1834, MacNish described sleep as a stage of unconsciousness: “the intermediate stage between wakefulness and death (as cited in Carskadon & Dement, 2005, p. 9). More modern definitions describe sleep as “a reversible behavioral state of perceptual disengagement from and unresponsiveness to the environment” (Carskadon & Dement, 2005, p. 16). Similar to the dichotomous view of human wakefulness and sleep, the state of sleep can also be conceptualized as occurring in two overarching stages: rapid eye movement (REM) and non-REM (Iber, Ancoli-Israel, Chesson, & Quan, 2007). Non-REM includes three stages: non-REM stage 1 (N1), non-REM stage 2 (N2), and non-REM stage 3 (N3), or slow wave sleep (SWS), which was formerly known as stage 3 and stage 4 sleep (Iber et al., 2007). All stages of sleep serve specific functions and are easily differentiated through the use of an electroencephalogram (EEG) and other measures. These stages occur in cycles during the night—the time of day in which the average human body is conditioned to sleep. These sleep patterns can vary in the presence of sleep disorders and during aging (Carskadon & Dement, 2005).

N1 is a transition state between wakefulness and sleep, during which drowsiness is present, muscle activity slows, and the desire or drive to sleep increases (Carskadon & Dement, 2005). During N1, an EEG shows moderate-amplitude theta waves (brain waves that are 4 to 8 Hz; Léger et al., 2018). N2 is a period of light sleep, during which eye movement virtually stops, the heart rate slows, and body temperature decreases (Carskadon & Dement, 2005). This stage is distinct in that sleep spindles (events during

non-REM sleep with 12-14Hz waves that wax and wane every 0.5 to 5 seconds; Dijk, Hayes, & Czeisler, 1993; Loomis, E. N. Harvey, & Hobart, 1935) and k-complexes (high-voltage, high-amplitude biphasic wave patterns that are often associated with sleep spindles; Loomis, E. N. Harvey, & Hobart, 1938) appear and increase during the transition from N2 to N3/SWS (Léger et al., 2018). SWS is a stage of deep sleep that is characterized by EEG output of slow wave activity at less than 2 Hz (Iber et al., 2007). SWS has been linked to memory consolidation, specifically declarative memory. When SWS is reduced, individuals are likely to experience difficulties in maintaining attention, decision-making, and working memory during wakefulness. They may also experience a decline in mood and increase in daytime sleepiness (Léger et al., 2018).

REM is the stage of sleep that occurs most during the second half of the night and is characterized primarily by dreaming and the rapid eye movements that are noticeable under the eyelids and measurable by electrooculogram (EOG; Carskadon & Dement, 2005; Dijk, 2009; Somers et al., 2008). Additionally during REM, the body becomes paralyzed temporarily (as evidenced by greatly reduced electromyogram [EMG] activity, a tool used to measure muscle activity); heart rate increases (as evidenced by electrocardiogram [ECG], which measures heart rhythm); blood pressure rises; breathing becomes more rapid, irregular, and shallow; and body temperature is not as well regulated (Somers et al., 2008). During both non-REM and REM sleep, sensitivity to temperature is greatly reduced; specifically, poikilothermia occurs, in which body temperature varies due to the environment, much like cold-blooded animals (Kamdar, Needham, & Collop, 2012). These stages of sleep comprise an individual's unique sleep architecture, which is subject to change with age (Ohayon et al., 2004).

Sleep Changes Associated with Aging

Researchers have sought to capture age-specific alterations across the human lifespan. Foley, Monjan, S. L. Brown, Simonsick, Wallace, and Blazer (1995) conducted an epidemiological study analyzing common sleep concerns in 9,217 participants, ages 65 and older. The five common sleep disturbances and symptoms of sleep-related daytime dysfunction for older adults were difficulty initiating sleep, difficulty maintaining sleep, early morning awakenings, needing to nap during the day, and not feeling rested upon waking. The participants were asked specific questions regarding these common concerns and replied on a Likert scale to identify frequencies of “rarely or never,” “sometimes,” or “most of the time” (Foley et al., 1995).

The researchers found that between 54% and 65% of these older participants reported that at least one of the five concerns was a “chronic condition,” which was not operationalized by the authors. Insomnia symptoms were significantly higher in female participants. Nevertheless, age was not found to be significantly associated with insomnia or not feeling rested. Instead, the researchers found that poorer health status and depression symptomology were significantly associated with higher frequency of overall sleep concerns (Foley et al., 1995).

Grandner, Martin, et al. (2012) also explored the prevalence of these sleep concerns across age groups using the 2006 Behavioral Risk Factor Surveillance System, an annual telephone interview survey of adults over the age of 18 across the U.S. They analyzed results of the sub-surveys that measured SCD (Self-Reported Sleep Disturbance) and for tiredness (Self-Reported Tiredness/Lack of Energy), thus assessing the severity of SCD and a symptom of sleep-related daytime dysfunction (tiredness). For

both measures, women reported more sleep disturbances and more somnolence than men. In regard to aging, Grandner, Martin, et al. found curvilinear relationships between both sleep disturbance and age and between tiredness and age. The researchers found that younger individuals (ages 18 to 24) were more likely to report tiredness than older individuals (ages 80 and older). The lowest reports of tiredness were observed in individuals 65 to 69 years of age (Grandner, Martin, et al., 2012).

The authors presented several possible explanations for their findings, positing a general correlation between overall health and sleep-related outcomes (Grandner, Martin, et al., 2012). Specifically, they postulated that individuals with very poor health are not likely to survive to older age and that those who do survive have developed a degree of resilience to further health decline. Another possible explanation was that the perception of “acceptable health status” is heavily influenced by age-related changes. As such, older adults’ assumptions of “normal” health for their age may have been adapted to fit their lowered expectation threshold for good health. As such, older cohorts may be less likely than younger cohorts to report SCD and tiredness because older adults may view these experiences as a normal part of aging (Grandner, Martin, et al., 2012).

To assess this further, researchers evaluated specific SCD differences among 592 normal sleepers between the ages of 20 and 96 years, evaluating SL, NWAK, WASO, and TST by collecting 2 weeks of sleep diaries (Dillon et al., 2014). From this data, they found that SCD varied individually within each age group; however, some age groups showed less within-group variability for specific SCDs. Specifically, TST and NWAK became less variable within older age groups (65 to 96 years; Dillon et al., 2014). Furthermore, the researchers found that the variability of TST between age groups was

impacted by demographic variables, such as sex and race (Dillon et al., 2014).

Conversely, the present controlled for these variables by matching the different age groups by sex, race, and body mass index (BMI). Although Dillon et al. (2014) appeared to have extensively assessed SCD across age groups, the researchers did not assess for EMA, sleep-related daytime dysfunction, or whether the participants considered their SCDs to be problematic.

There is some evidence to suggest that subjective age may have more of an impact on what individuals perceive as “normal” or acceptable aging than chronological age. Stephen and colleagues (2017) defined subjective age as how old an individual feels and specified that this may differ from the individual’s chronological age. In their study, they found that older reported subjective age was associated with an increased likelihood of sleep difficulties. As such, the researchers suggested that subjective age can be a biopsychosocial marker of aging, as it can be a more accurate marker of an individual’s risk for worsened sleep quality (Stephen et al., 2017). Regardless of chronological or subjective age, there is substantial evidence that changes in sleep and sleep continuity for aging individuals may result from developmental changes in sleep architecture.

To evaluate architectural sleep changes in aging, Ohayon, Carskadon, Guilleminault, and Vitiello (2004) conducted a comprehensive meta-analysis examining 65 studies between 1960 and 2003. These studies included 3,577 individuals between the ages of 5 and 102 years, whose sleep patterns were evaluated objectively with polysomnography (PSG) or actigraphy, two conventional methods of measuring sleep. The researchers examined overall age-related trends by plotting the mean values of the following objective sleep variables: TST (time spent asleep during the night); sleep

efficiency (SE%; amount of time in bed spent sleeping divided by the total amount of time in bed); SL (length of time to fall asleep); WASO (time spent awake in the middle of the night after sleep onset and before final wake time); percentages of S1, S2, and SWS; percentage of REM sleep during TST; and REM latency (amount of time that passes between sleep onset and first REM cycle; Ohayon et al., 2004)

Ohayon et al. (2004) found that, within the age trends for adults, the sleep variables of TST, SE%, percentage of SWS, percentage of REM, and REM latency were all negatively correlated with age, thus indicating an age-related decline in these variables, associated with normal sleep and health. Specifically, TST decreased roughly 10 minutes and SWS decreased approximately 2% with each aging decade. SE% did not show a significant decrease until 40 years old, after which there was a 3% decrease in each subsequent decade. Percentage of REM was compared between young adults and adults 60 years or older to show a 4% decline in the older group.

Although the variables mentioned earlier decreased with age, the researchers found that other variables increased, namely SL, percent of S1, percent of S2, and WASO (Ohayon et al., 2004). The researchers defined percentages of S1 and S2 as the amounts of time within total sleep time the individual spent within each of these stages of sleep (Ohayon et al., 2004). Specifically, percentages of S1 and S2 each increased by 5% between ages 20 and 70 years. Percentage of REM decreased but ultimately remained unchanged after the age of 60. Conversely, other studies within the meta-analysis found that REM began to decline again after 60 years of age (Ohayon et al., 2004). Unfortunately, the authors did not report how this decrease in percentage of REM impacted sleep quality or symptoms of sleep-related daytime dysfunction. Furthermore,

the researchers also did not assess whether the participants endorsed these changes in sleep to be problems.

Dijk, Groeger, Stanley, and Deacon (2010) also analyzed age-related changes in sleep architecture, specifically in SWS, and how these architectural changes related to daytime sleepiness in older individuals. The researchers recruited 110 healthy volunteers, stratifying them into age groups of 20 to 30 years, 40 to 55 years, and 66 to 83 years. These participants were assessed using the Multiple Sleep Latency Test (MSLT) and Karolinska Sleepiness Scale (KSS) to obtain objective and subjective sleep data (Dijk, Groeger, Stanley, & Deacon, 2010). The researchers found that healthy aging was associated with a reduction in daytime sleepiness, sleep continuity, and SWS and an increase in SCD, such as difficulty falling asleep, maintaining sleep, and with waking up earlier than intended. The authors theorized that this change in sleep architecture might be due to a lessening need for sleep in healthy adults without sleep disorders (Dijk et al., 2010). The study focused primarily on the measurement of sleep continuity through subjective and objective measures. The subjective measures within this study included the KSS and the Pittsburgh Sleep Quality Index (PSQI), and the objective measures included PSG and actigraphy. The study examined SCD and associated excessive daytime sleepiness (EDS); however, the researchers assumed that the existence of SCD constituted a perceived problem. They evaluated a causal relationship between these two variables using the data collected after implementing SWS disruption for half of the participants. The individuals who experienced this SWS disruption reported significantly higher scores on the KSS, a subjective measure of daytime sleepiness (Dijk et al., 2010).

Retirement effect. In addition to tiredness, fatigue is also considered a symptom

of daytime dysfunction that research suggests increases with age. Åkerstedt, Discacciati, Miley- Åkerstedt, and Westerlund (2018) collected sleep and fatigue data from 8,159 participants between the ages of 18 and 68 over the course of 8 years. The researchers collected sleep data using basic sleep diaries and the KSS. As opposed to much of the research in the field, these researchers appear to have assessed SCD severity and reported sleep-related daytime dysfunction.

Additionally, the researchers measured fatigue using a single item measured using a Likert scale ranging from 1 (not at all) to 5 (very much), and found that reported symptoms of daytime dysfunction increased for all age groups except the oldest age group (57 to 68 years). Overall, the sleep results supported the findings by Dijk et al. (2010), in that overall sleepiness was lowest in older participants when compared to younger participants. In regard to fatigue, there was decrease over time, with the lowest reported fatigue in the oldest group. The researchers posited that this is likely due to a strong retirement effect, as most of the older group was retired. This retirement effect is a phenomenon in which individuals experience positive changes following retirement, such as reduced fatigue and reduced sleep disturbances (Åkerstedt, Discacciati, Miley- Åkerstedt, & Westerlund, 2018). The researchers did not directly evaluate a causal relationship between SCD and symptoms of sleep-related daytime dysfunction; however, they observed several trajectories across the 8-year period in which there was a significant decrease in fatigue and a simultaneous significant increase in sleep duration (Åkerstedt et al., 2018).

In a longitudinal study in France, Vahtera et al. (2009) analyzed the retirement effect and its impact on SCDs in French adults at least 7 years pre- and post-retirement

age, which the authors defined as age 55, as this is the youngest average age of retirement in France. The authors focused on measuring SCDs in 14,714 employees between the ages of 37 and 63. They assessed SCDs using a single-item measure, asking whether the participant had experienced SCD within the past 12 months. Other measures used related only to potential medical conditions. The researchers found that individuals experienced fewer SCDs following retirement unless medical conditions were endorsed as the reason for retirement; however, only 4% of the participants reported medical conditions as their reasons for retirement (Vahtera et al., 2009).

Based on these results of Vahtera et al.'s (2009) study, it is difficult to determine whether quality of sleep changed and at what part of the night the participants were experiencing SCDs. Furthermore, the authors did not appear to operationally define SCDs for the participants. As with many country-specific studies, caution may be prudent in generalizing findings. Finally, another concern is the use of a single-item measure, which is associated with several potentially fatal psychometric limitations. These psychometric limitations include the inability to measure internal consistency (Wanous & Reichers, 1996) and the general lack of content validity (Van Hooff, Geurts, Kompier, & Taris, 2007). As such, these results should be interpreted with caution.

Limitations aside, it is evident that sleep patterns change across the human lifespan. Garland, Rowe, Repa, Fowler, Zhou, and Grandner (2018) further evaluated this change over 10 years in individuals between the ages of 20 and 80 years. They sought to compare insomnia symptom prevalence in Canadian provinces between the years of 2002 and 2012. The researchers assessed sleep using a single-item question: "How often do you have trouble going to or staying asleep?" The participants answered

on a Likert scale ranging from 1 (none of the time) to 5 (all of the time). The researchers then categorized individuals who reported scores of 4 (most of the time) or 5 (all of the time) as “poor sleepers.” Although the researchers found that the prevalence of insomnia symptoms increased over the 10-year period, they indicated that the increase, 1.5%, was modest. The authors found that the increase in insomnia symptoms over this 10-year period was due to the subsample of women between the ages of 40 and 59 years. The researchers indicated that changes in this group might be attributable to hormonal changes that occur during menopause (Garland et al., 2018).

It is possible that the increase of insomnia symptoms does not reflect actual change, because with single-item measures such as the unvalidated one used in Garland et al.’s (2018) and Vahtera et al.’s (2009) studies, it is difficult to determine whether this is a valid measure of SCD. Additionally, the use of a single-item measure of insomnia offers the same psychometric limitations listed in Vahtera et al. (2009). Moreover, the single-item does not capture the length of SL, WASO, or TST. It also may not capture EMA, which is more prevalent in older adults (Ohayon et al., 2004). Furthermore, although individuals may endorse difficulty falling or staying asleep, they were not asked whether this affects their daytime functioning or whether they consider these SCDs to be problems, defined for our purposes as a problem endorsement.

SCD maintaining factors. As sleep worsens, individuals may engage in maladaptive compensatory strategies to offset the negative consequences of reduced TST, which can perpetuate insomnia (A. G. Harvey, 2002b). One such behavior that is frequently seen in adults is napping. Foley, Vitiello, Bliwise, Ancoli-Israel, Monjan, and Walsh (2007) used the National Sleep Foundation’s 2003 Sleep in America Poll to assess

nap frequency, sleep quality, and EDS in 1,497 individuals between the ages of 55 and 84 years. The researchers found that 15% of the 1,497 participants ($n = 224$) reported regular napping, which was defined as napping 4 to 7 times per week. Of these 224 participants who reported regular napping, 10% were between the ages of 55 and 64 and 25% were between the ages of 75 and 84. These regular nappers appeared to differ significantly from non-regular nappers. Specifically, regular nappers worked fewer hours, had higher BMIs, and endorsed more comorbid conditions and symptoms, namely physical pain, nocturia, memory complaints, and higher rates of depression.

Regarding sleep-specific symptoms, the regular nappers subjectively reported poor to fair sleep quality, a TST of less than 6 hours each night, and difficulty falling and staying asleep, and waking up too early. Despite these endorsements, there was no relationship between regular napping and diagnoses of sleep disorders, including insomnia (Foley et al., 2007). Although the authors assessed napping prevalence concerning age, sleep quality, and EDS—an element that was not evaluated in this study—it is difficult to determine the severity of the participants' poor sleep quality as the measure did not include questions about SL, WASO, or EMA.

Although Foley et al. (2007) did not find a relationship between regular napping and insomnia, Ancoli-Israel and Martin (2006) illustrated in their meta-analysis some inconsistencies in the relationship between SCD and napping. For instance, although insomnia can lead to daytime sleepiness and daytime sleepiness can lead to napping, the relationship between insomnia and napping is unclear. The researchers posited that additional research must be conducted in order to determine a causal relationship. These studies would need to include measures of SCD (SL, WASO, EMA, TST) as well as

measures of sleep-related daytime dysfunction and subjective sleepiness that lead to the decision to engage in napping behaviors (Ancoli-Israel & Martin, 2006).

Interestingly, researchers found that daytime napping for less than 1 hour in individuals ages 65 and older could be potentially beneficial (Keage et al., 2012). The researchers collected sleep data and Mini-Mental Status Examination (MMSE) scores from individuals between the ages of 64 and 95 years. Based on their baseline data, individuals were placed into one of two groups: the higher MMSE group consisted of individuals who obtained MMSE scores of 26 to 30, and the lower MMSE group consisted of individuals who obtained MMSE scores of 22 to 25. Individuals who had MMSE scores of less than or equal to 21 were excluded from the study, as the researchers identified this score as indicative of cognitive impairment. Keage et al. (2012) found that individuals who napped regularly for less than 1 hour per day were less likely to be in the lower MMSE group and showed less daytime cognitive impairment. Of note, the protective factor obtained through napping did not have the same effect for individuals who napped for over 1 hour daily (Keage et al., 2012).

Cognitive decline. Several studies evaluated sleep in the context of cognitive decline (Schmutte et al., 2007). Schmutte et al. (2007) assessed SCDs in 375 adults between the ages of 75 and 85 who did not meet criteria for dementia and hypothesized that SCDs would be associated with reduced cognitive functioning. They collected sleep data through a 54-item sleep questionnaire, which measured SL, TST, and NWAK, and asked a single question regarding insomnia: Do you have insomnia or trouble sleeping? The researchers found that 22.7% of the participants endorsed insomnia or “trouble sleeping” (Schmutte et al., 2007). The researchers presented some limitations regarding

their method of data collection: they did not differentiate between SL and WASO and they did not specify a timeframe for when the participants had to experience these SCDs or sleep-related daytime dysfunction.

Waller et al. (2016) also compared sleep quality and EDS in cognitively impaired and cognitively unimpaired adult males who were, on average, 57 years of age. The researchers hypothesized that decreased sleep quality and increased daytime sleepiness would be more prevalent in cognitively impaired individuals ($n = 92$) when compared to cognitively unimpaired individuals ($n = 97$). The researchers evaluated sleep quality and EDS questions from the Pittsburgh Sleep Quality Index (PSQI) and the Epworth Sleepiness Scale (ESS). Results indicated a decline in cognition was related to a decrease in sleep quality, as evidenced by slightly significant differences in PSQI scores between cognitively intact (4.31 ± 2.32) and cognitively impaired (5.29 ± 3.70) individuals (Waller et al., 2016). There were no group differences in EDS, SCD (e.g., difficulty falling asleep, staying asleep, or waking up earlier than intended), or SL (Waller et al., 2016).

The aforementioned study appears to measure primarily sleep-related daytime dysfunction and does not consider the severity of these SCDs. A measure of SL, WASO, EMA, and TST would have more fully informed this decrease in sleep quality for middle-age to older males with cognitive impairment (Waller et al., 2016). It is evident from these studies and others that the aging and sleep literature mainly report on severity of SCD or sleep-related daytime dysfunction but rarely measure both. Furthermore, researchers make the assumption that endorsing the existence of SCD and/or sleep-related daytime dysfunction constitutes a problem. Additional categorization of the sleep

literature will be presented to illustrate this discrepancy further. See Appendix A for illustration of the categorical breakdown of cited research.

Insomnia Disorder

Diagnosis. Historically, in both academia and clinical practice, insomnia was perennially considered to be a supplementary symptom of other presenting conditions, such as major depressive disorder (MDD), anxiety, chronic pain, and diabetes. When insomnia existed in the presence of another condition, it was considered to be secondary insomnia. It was only when the insomnia appeared to exist on its own that it was considered to be primary insomnia (M. D. Mitchell, Gehrman, Perlis, & Umscheid, 2012). The *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition-Text Revision (DSM-IV-TR)* listed the diagnostic criteria for primary insomnia as “difficulty initiating or maintaining sleep, or nonrestorative sleep, for at least 1 month” (APA, 2000, p. 599). Moreover, the criteria stated that, in order to receive a diagnosis of primary insomnia, the sleep disturbances should not occur within the presence of other sleep disorders and mental disorders.

Insomnia disorder was eventually recognized as a primary diagnosis due to mounting evidence that the symptom pattern often presents in the absence of other, “primary” mental or physical disorders, and treating those primary disorders does not always lead to insomnia symptom abatement as well (APA, 2013). Indeed, such coincidental remission was not always supported and insomnia frequently presents in the absence of another “primary” disorder (Morgenthaler et al., 2006). This change is reflected in fifth edition of the *DSM (DSM-5)*.

Prevalence. Insomnia disorder prevalence has been widely studied; however, the prevalence rates vary by population, assessment measure, and how the researchers define insomnia (Morin & Jarrin, 2013). For example, according to the *DSM-5*, as many as one third of the adult population have experienced insomnia symptoms; however only 6% to 10% have met criteria for an insomnia disorder diagnosis (APA, 2013). Generally, insomnia symptoms have a global prevalence of 30% to 35% (Morin et al., 2015). The actual reported prevalence of insomnia disorder ranges from 3.9% to 22.1%, depending on the country and the basis for diagnostic criteria, which can vary based on diagnostic guidelines (K. F. Chung et al., 2015).

DSM-5 insomnia criteria. The APA's (2013) *DSM-5* describes insomnia disorder as "a predominant complaint of dissatisfaction with sleep quantity or sleep quality" (p. 362). Insomnia disorder, which is considered the most prevalent sleep disorder, is usually associated with difficulty falling asleep (initial insomnia), difficulty staying asleep (middle insomnia), and waking up earlier than intended (late insomnia). To meet criteria for an insomnia disorder diagnosis, the individual must experience significant daytime impairment in critical areas of functioning (e.g., social, occupational, educational, neurological, behavioral, and cognitive), symptoms must occur at least 3 nights each week, and symptoms must be present for at least 3 months. The sleep disturbances and daytime sequelae must exist despite ample sleep opportunity, to differentiate the disorder from insufficient sleep due to other causes (APA, 2013).

Furthermore, the SCD is not better explained by the physiological effects of substances or by another sleep-wake disorder, mental disorder, or medical disorder, which can be co-occurring (APA, 2013). Mental health professionals should specify

whether the disorder co-occurs with another sleep-wake disorder, a medical disorder, or a psychological disorder. Additionally, mental health professionals should determine the duration of the disorder by indicating whether insomnia is episodic (symptoms occur between 1 to 3 months), persistent (symptoms are present for longer than 3 months), or recurrent (two or more episodes of insomnia occur within 1 year; APA, 2013). If the insomnia is acute (symptoms are present for less than 3 months), the disorder should be coded as “other specified insomnia disorder” (APA, 2013).

ICSD-3 insomnia criteria. According to the *International Classification of Sleep Disorders, 3rd Edition (ICSD-3)*, chronic insomnia disorder occurs when a patient or caregiver reports difficulty falling asleep or staying asleep, or waking up earlier than intended. A primary difference between the *ICSD-3* and the *DSM-5* is that the former also includes criteria for children specifying that chronic insomnia also occurs if the child shows resistance to going to bed at an “appropriate” time or difficulty sleeping without the help of a caregiver (American Academy of Sleep Medicine [AASM], 2014).

The ICSD-3 describes both nighttime and daytime difficulties associated with insomnia disorder. Related to daytime difficulties, the ICSD-3 describes how patients may exhibit fatigue; impaired attention, concentration, or memory; decreased performance in different situations; irritability; daytime sleepiness; behavioral problems such as impulsivity or aggression; reduced motivation; decreased energy; susceptibility to errors or accidents; and expressed concerns or dissatisfaction with sleep (AASM, 2014; Ohayon, 2002). Daytime difficulties cannot be explained by lack of sleep opportunity or a poor sleep environment. SCDs must be present when the patient has enough time for sleep and when the individual’s sleep environment is safe, without distractions, dark,

quiet, and comfortable (AASM, 2014). These concerns must be present three times a week for over 3 months. If symptoms were present for less than 3 months, then the individual would not qualify for a diagnosis of chronic insomnia. Instead, they would receive a short-term insomnia disorder diagnosis. This diagnosis is similar to its chronic counterpart, except that the symptoms have not met the 3-month threshold necessary for a diagnosis of chronic insomnia disorder. Finally, this diagnosis is given when these symptoms are not better explained by another sleep disorder (AASM, 2014; Ohayon, 2002).

The likelihood of individuals meeting criteria for an insomnia disorder diagnosis increases for those who are female, individuals in older adulthood, and those with a lower socioeconomic status (SES; APA, 2013; Ancoli-Israel & Roth, 1999). Research indicates that African American women are the most likely group to experience insomnia at some point in their lives (Grandner et al., 2010). Furthermore, individuals with insomnia are also likely to have a comorbid diagnosis of MDD (Grandner, Martin, et al., 2012).

Models of Insomnia

Sleep drive. Sleep-wake regulation is best described using a two-process model: the circadian process (Process C) and the homeostatic sleep process (Process Sleep Homeostasis [Process S]). These processes interact to determine sleep onset and waking behaviors (Borbély, 1982). Process C is the alerting signal portion of the model that is controlled by the circadian clock and is heavily influenced exogenously by the environment, mainly by light (a zeitgeber or time-giver; Borbély, 1982). This circadian clock exists in humans and most mammals and controls many physiological processes, such as body temperature. Process C also influences many aspects of alertness. The

endogenous-influenced circadian rhythm is regulated by the suprachiasmatic nucleus (SCN). The SCN, which is located in the anterior hypothalamus, controls sleep and wake behaviors across an approximate internal 24-hour period (Achermann, 2004).

Alternatively, Process S is related to sleep homeostasis (Borbély, 1982). While an individual is awake, sleep pressure builds with the accumulation of adenosine, which increases the propensity for sleep. This sleep pressure may directly oppose the circadian alerting signal (Process C). Once the individual falls asleep, that sleep pressure and adenosine reduce significantly. These two opposing processes, Process C and Process S, explain how individuals can maintain wakefulness even as sleep pressure builds. This model creates a regular pattern of sleep propensity (Borbély, Daan, Wirz-Justice, & Deboer, 2016).

Most individuals fall asleep at the beginning of the night because of the influence of both Process S and Process C. These two processes are synchronized in individuals who sleep well. For individuals who sleep well, sleep pressure and adenosine are highest in the evening (Process S), having significantly increased throughout the day, and these individuals will typically stay asleep during the night. This pattern is also influenced by a low circadian alerting signal (Process C) and melatonin levels. Conversely, individuals may wake in the morning because of reductions in sleep pressure, adenosine, and melatonin. Most individuals can stay awake throughout the day despite sleep pressure build up because the circadian alerting signal is high at these times of low to moderate adenosine and melatonin (Borbély, 1982; Borbély et al., 2016). If one is awake for an extended period of time, sleep pressure continues to build until sleep modifies the two systems (Borbély et al., 2016). Although the two-process model can allow for a

relatively regular sleep-wake schedule, stressors can interfere with sleep and lead to adverse effects.

Spielman's 3P model. In the field of behavioral sleep medicine, the most widely-accepted model to explain the natural progression of insomnia disorder is known as Spielman's 3P model, which is illustrated by the three Ps of predisposing, precipitating, and perpetuating factors that lead to the manifestation of insomnia disorder. A predisposition in itself will not directly lead to insomnia (Spielman, Caruso, & Glovinsky, 1987). Predisposing factors include a biological predisposition to be "good" or "bad" sleepers or to develop conditions that could affect sleep. These predisposing factors can include family history of insomnia, high arousability (easily awoken), previous episodes of insomnia, mental health difficulties (e.g., depression or anxiety), and bodily pain or discomfort. Other predisposing factors include higher BMI, heavy alcohol consumption, and cigarette smoking (LeBlanc et al., 2009).

Precipitating factors or triggers for insomnia usually involve stressors, which can cause disruptions in sleep for a short period. These stressors can include environmental, social, familial, occupation, financial, or medical stressors. In those who do not develop insomnia, once these acute stressors subside, sleep can return to baseline. That is, sleep will return to baseline unless perpetuating factors are at play. If sleeplessness persists, individuals are likely to implement maladaptive coping strategies that can worsen their sleep and cause them to transition from acute insomnia to chronic insomnia (Spielman et al., 1987). These maladaptive coping strategies include but are not limited to extending the sleep opportunity and time in bed by going to bed earlier and getting out of bed later, napping behaviors, engaging in non-sleep-related behaviors in bed, and consuming

caffeine at unpropitious times. Although the intention behind these strategies is to combat insomnia, when maladaptive, these behaviors lead to the worsening of the sleep. In actuality, this can lead to dysfunctional beliefs and attitudes surrounding sleep, including those related to depression, anxiety, frustration, and irritability (Spielman et al., 1987).

Perlis's 4P Model

Perlis, Ellis, Kloss, and Riemann (2016) extended Spielman's model with the addition of a fourth P: Pavlovian conditioning. This addition considers classical or Pavlovian conditioning as a perpetuating factor and seeks to further inform the course of insomnia disorder and related treatment modalities, specifically cognitive behavioral therapy for insomnia (CBT-I; Perlis, Jungquist, M. T. Smith, & Posner, 2006). Pavlovian conditioning refers to a method in which an organism develops a conditioned response through the process of (often) repeated presentations of a specific, unconditioned stimulus in the presence of another salient stimulus. Concerning insomnia disorder, this means that, although a stimulus (such as the bed) once elicited a sleep response, it can become associated, in poor sleepers, with wakefulness, frustration, irritation, and anxiety after repeated exposures of the bed in a wakeful, frustrated, irritated, or anxious state. This conditioned response serves to perpetuate insomnia disorder and exacerbate daytime dysfunction (Perlis, Ellis, et al., 2016).

Differential Diagnoses of Select Sleep Disorders

Just as living cells are prone to malformation, human sleep is prone to maladaptive variations, which can lead to symptoms of various sleep disorders. For example, some of these disorders include sleep apnea, restless leg syndrome/periodic

limb movement disorder, circadian rhythm disorders, and shift work disorder (AASM, 2014; APA, 2013). Although these do not encompass all existing sleep-wake disorders, they are the primary differential diagnoses assessed in the online questionnaire used in the present study.

Obstructive sleep apnea-hypopnea syndrome. Obstructive sleep apnea-hypopnea syndrome (OSAHS) is the most common breathing-related sleep-wake disorder and is characterized by upper (pharyngeal) airway obstruction during sleep (APA, 2013). “Apnea refers to complete obstruction or cessation of airflow, whereas “hypopnea refers to a cessation of airflow to less than 50% of the normal flow during sleep, without complete obstruction (APA, 2013). Both are associated with oxygen desaturation, which is a reduction in oxyhemoglobin saturation (Somers et al., 2008). To meet criteria for apnea or hypopnea events, this reduction in normal breathing must last 10 seconds or more, making it difficult for oxygen to travel to the brain and other organs (APA, 2013). This can lead to snoring, gasping during sleep, and hypersomnolence (EDS), and can increase the risk for a morning headache, behavioral changes, hypertension, diabetes, sexual dysfunction (mainly in men), cancer, and dementia (Somers et al., 2008). Men, especially adult men between the ages of 40 and 60, are at highest risk for developing OSAHS. This is especially true if they are obese ($BMI > 30$) or have a large neck circumference, with the latter being the most significant predictor of OSAHS (Somers et al., 2008). OSAHS is evaluated and diagnosed using PSG. Particular attention is paid to the apnea-hypopnea index (AHI), which records how many apnea/hypopnea “events” an individual has per hour, ultimately diagnosing the severity of the disorder (Somers et al., 2008).

A recent study observed age factors significantly associated with OSAHS in a group of 90 individuals (Hongyo et al., 2017). These individuals were separated into non-elderly (< 65 years) and elderly (\geq 65 years) groups and further categorized as having mild to moderate OSA or severe OSA. For the purposes of this study, the researchers defined mild to moderate OSA as having an AHI between 5 and 30 and severe OSA as having an AHI greater than or equal to 30. Researchers found that greater BMI was significantly correlated with a greater AHI for both age groups. Age was associated with AHI only in the elderly group, independent of BMI (Hongyo et al., 2017).

Restless leg syndrome. Restless leg syndrome (RLS) is a sensorimotor, neurological sleep-wake disorder and is primarily distinguished by the urge to move one's legs (APA, 2013). Roughly 10% of American adults suffer from RLS. Within this percentage, 5 million American adults suffer from a moderate to severe form of the disorder (Allen et al., 2005). Although the disorder mostly affects adults, approximately 1 million school-age children in American also suffer from RLS (APA, 2013).

RLS is classically characterized by the intense urge to move one's legs, usually in response to uncomfortable or unpleasant sensations in the lower legs. These sensations of discomfort, or sensory disturbances, appear and worsen during periods of inactivity and are only relieved, temporarily, when individuals move their legs. RLS is primarily considered a sleep-wake disorder due to the worsening of symptoms during the evening or at night and the fact that the symptoms negatively impact sleep (APA, 2013). Because of the discomfort, timing of the onset of symptoms, reduced depth of sleep, and extreme urge to become active as a way of temporarily relieving symptoms, RLS can negatively

impact sufferers' sleep, causing daytime fatigue, impaired daytime functioning, and lowered quality of life (APA, 2013).

Trained clinicians attend to four basic complaints by the patients before diagnosing this disorder. First, patients must describe an overwhelming urge to move the affected limb or limbs. Second, patients will usually state that they experience discomfort if they do not move the limbs. The movement will usually relieve the discomfort for as long as they continue to move the limb. Third, the symptoms are usually triggered during times of rest, relaxation, or sleep. Finally, the symptoms worsen at night and/or may be absent during morning hours (Hening, 2004; Trenkwalder & Paulus, 2004). These limb movements can be further assessed using EMG during a PSG (Freedman & Roehrs, 2004).

Accurate diagnosis may include a neurological and physical exam, an in-depth assessment of the patient's medical and family history, current medications, and a series of laboratory tests to rule out other conditions (Hening, 2004). Accurate diagnosis of the disorder usually requires laboratory sleep studies that utilize PSG (Stefansson et al., 2007). Once manifested, RLS is a lifelong disorder for which there is no cure. The treatments and therapies available target the discomfort and symptoms associated with the disorder in order to improve the individual's overall quality of life (Trenkwalder & Paulus, 2004). Symptoms may worsen with age, but the increase in severity of symptoms appears at different rates in each individual (APA, 2013).

Individuals who seek treatment may experience misdiagnosis by clinicians who attribute the symptoms to nervousness, insomnia, stress, arthritis, muscle cramps, aging (Allen et al., 2005). If RLS is left untreated, the condition may cause exhaustion,

daytime fatigue, lower overall quality of life, and all of the symptoms associated with sleep deprivation (Hening, 2004). Individuals who suffer from RLS describe the condition and subsequent exhaustion as significantly affecting their occupations, personal relationships, and daily activities (APA, 2013). Without treatment, it is possible for individuals with RLS to experience a reprieve of symptoms for as long as a few months; however, this is only possible during the early stages of the disorder. Regardless of this occurrence, symptoms inevitably worsen over time if left untreated (Hening, 2004). Treatments include lifestyle changes and medication management.

Some lifestyle changes may help lessen the severity of symptoms. Examples of lifestyle changes include decreasing caffeine, alcohol, and tobacco (Allen et al., 2005). Additionally, physicians may recommend adding supplements to correct deficiencies that may cause or worsen RLS symptoms, such as lack of sufficient iron, folate, and magnesium. Physicians may also recommend changing poor sleep behaviors, increasing physical activity, massaging the legs, or treating the legs with ice or heat (Trenkwalder & Paulus, 2004).

Unfortunately, there is no single medication that will cure RLS; however, some medications exist that will help alleviate symptoms and improve quality of life. Research suggests that RLS is strongly associated with lower dopamine levels in the brain. The medications used to combat this symptom include dopamine agonists, which help activate the receptors that produce dopamine (Allen, 2004). Examples of these dopaminergic agents include reopinirole (brand name Requip) and ramipexole (brand name Mirapex). Physicians recommend patients take these medications within 1 to 2 hours of bedtime to reduce sleep-associated RLS symptoms (Allen, 2004). Other types of medications

prescribed to individuals with RLS include benzodiazepines, opioids, and anticonvulsants (Allen, 2004; Connor et al., 2011).

Periodic limb movement disorder. Periodic limb movements (PLMS) are described as “periodic episodes of repetitive, highly stereotyped limb movements that occur during sleep” (AASM, 2014, p. 293). As with OSAHS and RLS, PLMS can be documented using PSG, with a PLMS Index (frequency of movements per hour) compiled based on EMG data. If an adult experiences PLMS more than 15 times per hour, he or she meets the criteria for a diagnosis for periodic limb movement disorder (PLMD; AASM, 2014). Additionally, in order to meet this diagnosis, the individual must also experience sleep disturbances, decrease in sleep quality, or daytime impairment due to these PLMS (AASM, 2014).

Circadian rhythm sleep-wake disorders. As discussed previously, humans follow an endogenous circadian rhythm mandated by biology and an exogenous circadian rhythm controlled by the environment and societal requirements, such as an occupation (Achermann, 2004). Circadian rhythm sleep-wake disorders (CRSDs) occur when sleep disruptions result in a misalignment between endogenous and exogenous circadian rhythms. This misalignment can cause clinically significant distress leading to EDS and insomnia. In turn, this can also lead to impairment in social, occupational, or other critical areas of functioning (APA, 2013).

There are seven CRSDs addressed in the ICSD-3: delayed sleep-wake phase disorder, advanced sleep-wake phase disorder, irregular sleep-wake rhythm disorder, non-24-hour sleep-wake rhythm disorder, shift work sleep disorder, jet lag disorder, and circadian sleep-wake disorder not otherwise specified (AASM, 2014). Three of these

CRSDs are measured in the online questionnaire used in the present study: delayed sleep-wake phase disorder, advanced sleep-wake phase disorder, and shift work disorder. In delayed sleep phase type, individuals experience the inability to fall asleep or awaken at conventional times. As such, if given the opportunity, these individuals may get the proper amount of sleep but will fall asleep later and wake up later than desired. Alternatively, in advanced sleep-wake phase type, individuals may similarly get sufficient hours of sleep but experience difficulty maintaining wakefulness until a conventional bedtime and wake earlier than the environment generally demands (APA, 2013).

A third CRSD that explicitly affects workers with untraditional work schedules is shift work sleep disorder (APA, 2013). This disorder includes characteristics of insufficient sleep and excessive sleepiness. Shift work disorder affects individuals whose work hours occur during hours they would typically be asleep, thus impacting shift workers who work night shifts, rotating shifts, or extended hours that span multiple shifts (Basner et al., 2008). Approximately 20% of American workers work some form of shift work as part of their occupations. Shift work spans different vocations and is used as a way to keep countries competitive in the global market (Venkateshiah, Hoque, DelRosso, & Collop, 2017). Although this may be beneficial for a company and a country's overall economy, shift work can cause adverse outcomes for a worker's health and overall well-being (Gaba & Howard, 2002).

CRSDs—shift work type in particular—can significantly affect quality of life and health of workers. For instance, researchers assessed the shifts of registered nurses in hospital settings and found that half of their shifts were greater than 10.5 hours on

average (Rogers, Hwang, Scott, Alken, & Dinges, 2004). Additionally, 30.9% of the scheduled shifts were 12.5 hours; however, 38.7% of the shifts actually worked were of this length. The range of the overall reported shifts were 1.2 hours to 23.7 hours. Although only 6.8% of the shifts were mandatory overtime, nurses reported that for 143 shifts, they felt pressured to volunteer overtime. This presents many health concerns for the professionals and it can greatly impact patient care and the errors or near errors made by nurses (Rogers et al., 2004).

Rogers et al. (2004) found that the number of nursing errors was significantly affected by work duration, overtime, and number of hours worked per week. Specifically, the likelihood of a nurse making an error increased with longer work hours, and nurses' shifts could last up to 12.5 hours in length. Of the 199 patient errors and 213 near errors reported during the data collection of this study, over half of the errors (58%) and near errors (56%) involved medication administration. It is important to note a limitation of this study is that it is likely many errors or near errors were not documented by nurses. As such, the true number of errors resulting from extended work hours could be much greater than is reported, deleteriously affecting nurses, their patients, and institutions (Gaba & Howard, 2002).

Sleep disorders not only affect quality of life but can also increase a country's economic burden directly and indirectly. Hillman, Murphy, Antic, and Pezzullo (2006) evaluated the economic burden of sleep disorders in Australia in 2004. They found that the overall cost of sleep disorders in Australia's population of 20.1 million was \$7.4 billion, with only \$146 million allocated for treating the disorders. The remaining burden covered other aspects, such as comorbid conditions, work-related injuries resulting from

the disorders, non-work-related vehicle accidents, and productivity losses (Hillman, Murphy, Antic, & Pezzullo, 2006).

The sleep disorders mentioned above are not an exhaustive list, as there are still many others of importance, such as narcolepsy, REM behavior disorder, and central sleep apnea.

Sleep-Related Daytime Dysfunction Due to SCDs and Sleep Deprivation

Sleep deprivation. Sleep deprivation occurs when an individual is unable to obtain the necessary amount of sleep to function without impairment (Basner et al., 2008). Sleep deprivation can occur due to reduced sleep opportunity, insufficient sleep, or medical and psychiatric comorbidities (Lim & Dinges, 2008). Research suggests that factors on societal, social, and individual levels can ultimately lead to insufficient or inadequate quality of sleep. On a societal level, there is a heightened emphasis on technology, public policy, globalization, and the idea of a 24/7 society to overcome business differences in geography. On a social level, individuals are impacted by occurrences in the home or work involving race/ethnicity, socioeconomic status, religion, or culture. Finally, sleep can be affected on an individual level according to one's genetic predisposition, psychology, health, beliefs, behaviors, attitudes, and choices (Grandner, 2017).

Sleep deprivation can lead to adverse health outcomes and can impact general health, including cardiovascular health, metabolic health, mental health, immunologic health, and general performance. In some cases, individuals may experience more severe health conditions and possibly higher mortality rates. These detrimental effects on the

health of sleep-deprived individuals speak to the ethical, multicultural, and health policy concerns that are generally dismissed by employers (Grandner, 2017).

Not only does sleep deprivation lead to adverse health outcomes, but it can also result in a higher economic burden (Venkateshiah et al., 2017). Ultimately, sleep disturbances and deprivation can lead to a decrease in work productivity and performance and higher health care costs, which most likely relate to increases in medical and psychiatric disorders developed as a direct or indirect result of sleep deprivation (Hui & Grandner, 2015). These adverse outcomes usually occur when sleep deprivation becomes chronic. Chronic sleep deprivation can also lead to neurocognitive consequences, such as increasing the risk of drowsy driving and related accidents (Durmer & Dinges, 2005). A 2012 study assessed the proportion of crashes that involved drowsy drivers in a sample of 47,597 crashes between the years of 1999 and 2008. Of these crashes, drowsy driving was the primary cause for 7.0% of total crashes. Of those crashes, 13.1% of non-fatal crashes resulted in hospitalizations and 16.5% were fatal (Tefft, 2012). Finally, chronic sleep deprivation can also increase the risk of suicidality, and either lower the threshold for or exacerbate symptoms of medical and psychiatric comorbid conditions (Grandner, 2012; Perlis, Ellis, et al., 2016; Venkateshiah et al., 2017)

Neurocognitive consequences of poor sleep. According to Durmer and Dinges's 2005 study, when total sleep time was reduced to 4 to 6 hours per night for healthy adult participants between the ages of 21 and 64 years, cognitive performance declined significantly. This decline in cognitive performance increased over time as partial sleep deprivation continued. At a certain point, the level of impairment can appear

similar to impairment after 1 or 2 nights of *total* sleep deprivation. General lack of sleep (less than 6 hours) can result in neurocognitive deficits, such as diminished executive attention, working memory, and divergent higher cognitive functions (Durmer & Dinges, 2005), as well as physical consequences. Deficits in good sleepers can best be rectified if individuals return to a healthy, standard sleep routine, rather than attempting to “catch up” on sleep through sleep extension, napping, and other compensatory strategies (Perlis et al., 2006).

In some instances, such as while working, individuals may attempt to self-medicate with caffeine in an attempt to combat diurnal sleepiness and fatigue (Grandner, Knutson, Troxel, Hale, Jean-Louis, & Miller, 2014). Although this may help with alertness in the short-term, the substance does not protect against the aforementioned adverse effects of sleep loss. Subsequently, caffeine may mask the consequences of insufficient sleep and reduce motivation for change (Grandner et al., 2014).

Sleep and cognition. Sleep continuity disturbances and cognition are so interwoven that A. G. Harvey (2002a) created a cognitive model of insomnia. This model describes how dysfunctional cognitions and worry regarding sleep can lead to safety behaviors and, ultimately, cause insomnia maintenance or exacerbation. A. G. Harvey’s cognitive model of insomnia is based on the concept that individuals with insomnia experience worry about sleep that Morin (1993) described as unpleasant, intrusive thoughts that were particularly prevalent during the few hours before intended bedtime. Morin established that, for older adults in particular, individuals with chronic insomnia endorsed stronger and more dysfunctional beliefs and attitudes about their sleep when compared to same-age adults who were self-categorized as good sleepers. These

beliefs focused primarily on the perceived consequences of disturbed sleep rather than the poor sleep itself. Specifically, poor sleepers endorsed mostly hopelessness, in that they were afraid they would lose control, and feelings of helplessness, in that they feared the unpredictability of sleep and a lack of ability to ameliorate the issues. Although these cognitions were not the only ones present, they showed the biggest discrepancy between the good sleepers and the poor sleepers (Morin, 1993).

It should be noted that Morin (1993) found that poor sleepers experienced anxiety, worry, stress, hypervigilance, and obsessions regarding their sleep during daytime waking hours as well. As such, the foundation of the cognitive model is this harmful cognitive activity that is present both in bed and during waking hours, in which worries about sleep quantity and quality and its impact on daytime functioning are prominent. According to A. G. Harvey (2002a), these worries can, in turn, trigger arousal due to activation of the sympathetic nervous system (SNS), as well as emotional distress. The former is problematic in that activation of the SNS normally occurs in the presence of a real threat, where an individual would need to fight or flee. This directly impacts an individual's emotional state, likely increasing their emotional distress and reducing the propensity to sleep (A. G. Harvey, 2002a).

According to A. G. Harvey's (2002a) model, this arousal and distress can cause an individual to selectively attend to and monitor what he or she perceives to be sleep-related cues. This selective attention and monitoring can differ depending on the time of day. During the night, an individual may monitor their body for sensations that are both consistent with sleep (e.g., slowed heart rate) or inconsistent with sleep (e.g., racing heart beat), the environment for stimuli associated with wakefulness (e.g., noises around the

sleep environment), or the clock to either calculate how long it is taking to fall asleep or to calculate the remainder of their sleep opportunity. Upon waking, an individual may demonstrate confirmation bias and attend to their body for signs of poor sleep (e.g., heavy feeling and tiredness upon waking), and the clock to calculate sleep quantity (A. G. Harvey, 2002a).

Alternatively, during the day, the individual may attend to their level of daytime sleepiness; bodily sensations of fatigue or lethargy; any reduction in energy, memory, concentration, or an overall performance; and negative mood states, such as irritability or depression. Despite such monitoring, the individual will likely begin to experience some misperception regarding actual sleep quantity. He or she can underestimate the amount of sleep obtained. This misperception that he or she is sleeping less than he or she is can initiate or maintain distress and the use of safety behaviors regarding sleep (A. G. Harvey, 2002a).

Safety behaviors manifest in many medical or psychological disorders. A. G. Harvey (2002b) illustrated several safety behaviors. For example, when an individual believes that he or she will not sleep because their mind will not cease racing, the resulting safety behavior may be that the individual attempts to stop all thinking, only resulting in the exacerbation of all excessive cognitive activity. Even though this safety behavior has failed, the individual will still hold onto the dysfunctional belief that controlling their thoughts is the answer to obtaining better sleep (A. G. Harvey, 2002b). Other safety behaviors include trying to “catch up” on sleep, such as by napping or staying in bed longer, drinking excessive or poorly-timed caffeine to combat EDS, and changing sleep schedules to address sleep debt (A. G. Harvey, 2002b). Safety behaviors

for poor sleepers only serve to maintain or worsen an individual's insomnia and can occur during the night or the day.

An individual may also fear that he or she will not be able to function at their optimal level—or at all—with little sleep. As such, safety behaviors may involve reducing responsibilities or canceling appointments. Lessening productivity during the day only increases an individual's level of boredom and levels of daytime sleepiness (A. G. Harvey, 2002b). Ultimately, this allows more time for the individual to experience their preoccupation and other harmful cognitive activity regarding sleep. The belief that he or she must reduce productivity after a poor night of sleep is solidified, thus continuing the cycle. Without early intervention, an individual is likely to initiate, maintain, or worsen sleep deficits, which result in deficits in daytime performance and functioning (A. G. Harvey, 2002b). Negative cognitions can predate sleep difficulties, be associated with poor sleep, or co-occur with poor sleep, such as in the form of psychological comorbidities or even suicidality.

In one of the foundational articles that led to the development of A. G. Harvey's (2002a) cognitive model of insomnia, Morin, Stone, Trinkle, Mercer, and Remsberg (1993) examined negative sleep-related cognitions in older adults with ($n = 74$) and without ($n = 71$) endorsement of insomnia symptoms. The individuals who endorsed insomnia were seeking treatment for insomnia symptoms lasting longer than 6 months. Both groups were given the Dysfunctional Beliefs and Attitudes About Sleep Scale (DBAS). The researchers found significant differences between the individuals seeking treatment for insomnia and individuals who were considered good sleepers. First, there was a significant between-group difference regarding the perceived consequences of

insomnia in that the individuals with chronic insomnia were more likely to agree with statements that poor sleep would negatively impact their physical and mental health.

Good sleepers were more likely to disagree with these statements, indicating that those with insomnia were more likely to magnify the consequences of poor sleep.

Additionally, there were significant between-group differences regarding statements of control and predictability of sleep. Poor sleepers were more likely than good sleepers to endorse statements that reflected they worried about losing control over the ability to obtain adequate sleep (Morin et al., 1993).

Finally, the researchers found significant between-group differences in the construct of sleep expectations. One particularly interesting finding was that poor sleepers did not agree with the statement that an individual's sleep-need decreases with aging (Morin et al., 1993). This finding is relevant to the present study. It is evident that Morin et al. (1993) were assessing the category of sleep-related daytime dysfunction, specifically cognitive distortions; however, it would have been helpful if the authors had verified the diagnosis of insomnia within the participants seeking treatment and those who were identified as good sleepers. Understanding the severity of these sleep concerns would be beneficial in that it may illustrate some nuances in insomnia in general and in insomnia for older populations more specifically. For example, there may be individuals who categorize themselves as good sleepers but obtain similar sleep quantity and quality as individuals seeking treatment for insomnia. If these limitations were addressed, it would further inform Morin et al.'s (1993) point that cognition plays a fundamental role in treatment-seeking behavior.

Suicidality. Perlis, Grandner, Chakravorty, Bernert, G. K. Brown, and Thase (2016) conceptually outlined the progression of how a significant life stressor can lead to suicidality. Specifically, the researchers found that insomnia, poor sleep quality, and nightmares can significantly and independently predict and increase an individual's risk for suicidal ideation, suicide attempts, and death by suicide (Perlis, et al., 2016). This progression involves exposure of a significant life stressor, which then activates the hypothalamic-pituitary-adrenal (HPA) axis, thus mediating an individual's stress response. This activation can lead to mood disturbances (which would then result in even greater HPA axis activation) and SCDs due to insomnia or nightmares. These SCDs can likely lead to an individual being awake at night which can, in turn, lead to sleep loss and sleep inertia, the latter of which involves impaired cognitive and sensorimotor function (Perlis, Ellis, et al., 2016). From this, an individual can experience hypofrontality (state of decreased cerebral blood flow to the prefrontal cortex of the brain), impaired executive functioning, and altered higher cognitive functioning. When this wakefulness through long hours into the night is combined with mood disturbances, access to substances and weapons, and lack of access to social support, the individual's risk of suicidal ideation, suicide attempts, and death by suicide increases significantly (Perlis, Grandner, G. K. Brown, et al., 2016).

In a study supporting this model, Perlis et al. (2016) found that of 35,332 completed suicides in the U.S. for which time was documented, 63.9% occurred between midnight and 6 a.m. Suicide data were gathered using the National Violent Death Reporting System between 2003 and 2010. These numbers were compared to data from the American Time Use Survey, for which researchers specifically assessed percentage of

the American population awake for each hour of a 24- hour day (Perlis, Grandner, Chakravorty, et al., 2016).

Significantly, the researchers adjusted the data to reflect the percent of completed suicides compared to the estimated percent of the population awake at each time point (hourly intervals). During the midnight to 6 a.m. time period, the researchers estimated that at the lowest, 5.34% of Americans were awake (3 A.M.) and at the highest, approximately 47.40% of Americans were awake (6 A.M.). From these individuals expected awake, 63.9%, or roughly 22,577 (of 35,332), of completed suicides were documented as occurring between the hours of 12 a.m. and 6 a.m. In other words, suicides are 3.6 times higher between midnight and 6 a.m. than during what are considered waking hours. This illustrates the real life and death risks associated with poor sleep (Perlis, Grandner, Chakravorty, et al., 2016).

Assessment of Insomnia

Proper diagnosis of sleep disorders requires careful assessment. For insomnia disorder, these assessments fall into two categories: (a) subjective or self-report measures and (b) objective measures. There are far more self-report assessment measures than objective measures with regards to insomnia assessment. This is likely because self-report assessment measures are less expensive and take less time to administer (Ibáñez, Silva, & Cauli, 2018). Each variety of measure—both subjective and objective—assesses different aspects of sleep. As such, clinicians may choose to use a single measure or a combination of measures to evaluate presenting sleep concerns.

Self-report assessment measures. Diagnosis of insomnia disorder relies primarily on endorsements of difficulty falling asleep or staying asleep, or waking up too

early. Additionally, to meet criteria, there must be the presence of daytime dysfunction, which includes impaired decision-making abilities, irritability, and distress regarding current sleep (AASM, 2014; APA, 2013). As such, to meet diagnostic criteria, the individual must report experiencing significant daytime distress or impairment as a result of their insomnia symptoms (APA, 2013). Should a clinician choose to formally assess various aspects of sleep concerns, he or she may do so with a variety of measures, such as the ESS, the KSS, the DBAS, the PSQI, the Insomnia Severity Index (ISI), or the Sleep Disorders Symptom Checklist-25 (SCD-CL-25).

Epworth Sleepiness Scale. The purpose of the ESS is to differentiate patients with EDS from other patients with higher levels of alertness (Johns, 1991a). The scale has been validated for adult populations between the ages of 18 and 78 (Miletin & Hanley, 2003). This short questionnaire presents eight different situations and asks the patient to rate their likelihood of “dozing” in this situation on a scale of 0 (would never doze) to 3 (high chance of dozing). The patient’s total score represents their level of daytime sleepiness. Patients can be categorized as having Lower Normal Daytime Sleepiness (0-5), Higher Normal Daytime Sleepiness (6-10), Mild Excessive Daytime Sleepiness (11-12), Moderate Excessive Daytime Sleepiness (13-15), or Severe Excessive Daytime Sleepiness (16-24) (Johns, 1991a). Scores between 0-10 are considered normal; however, this is in the context of the general population and does not account for individuals with sleep disorders. Scores between 11-24 indicate EDS at a clinical level requires documentation in a clinical setting and may require additional action (Johns, 2000b). Patients who endorse Severe Excessive Daytime Sleepiness should be cautioned against driving or operating heavy machinery (Johns, 2000a). The ESS can

be used to assess daytime sleepiness levels over time in adult populations and is used in conjunction with behavioral sleep medicine interventions, such as during the sleep restriction phase of CBT-I (Johns, 1991b; Perlis et al., 2006). Of note, this scale does not take into consideration SCDs or other reasons for EDS. Furthermore, it does not ask the respondent if he or she considers their reported level of daytime sleepiness to be a problem.

Karolinska Sleepiness Scale. The KSS measures level of subjective sleepiness at a specific time point. This subjective sleepiness is evaluated on a 9-point scale with 1 being “extremely alert” and 9 being “extremely sleepy—fighting sleep.” A newer edition of the scale includes an additional option of 10, “extremely sleepy—falls asleep all the time.” Additionally, KSS scores are strongly correlated with the time of day the measure is administered and, as it is a measure of situational sleepiness, scores may fluctuate (Åkerstedt & Gillberg, 1990). Due to this sensitivity, the KSS is not used widely in clinical settings; however, it has been useful in assessing sleepiness in adults who face some level of circadian disruption, such as from jetlag or shift work (Kecklund & Åkerstedt, 1993).

Because this measure is used primarily to assess sleepiness fluctuations in response to different factors (e.g., drugs or environmental changes, in addition to the above) rather than consistent sleepiness due to non-circadian related sleep disorders, it is generally not used within clinical settings (Shahid, Wilkinson, Marcu, & Shapiro, 2012). Additionally, this measure only assesses one aspect of sleep-related daytime dysfunction (sleepiness) and does not consider other dysfunctions (such as difficulty concentrating), SCDs, or if the individual considers their sleepiness to be a problem.

Dysfunctional Beliefs and Attitudes About Sleep Scale. The DBAS was developed by Morin in 1993. This measure consists of 28 items and assesses five separate cognitive themes: amplification of consequences of insomnia, a feeling of loss of control regarding sleep, unrealistic expectations about sleep, misconceptions about causes of insomnia, and incorrect beliefs about sleep-related behaviors (Morin et al., 1993). The DBAS has been validated for adults between ages 55 and 88 (Morin et al., 1993), with a later study validating the use of the measure for 178 individuals with a mean age of 49.8 ($SD = 17.9$; Espie, Inglis, L. Harvey, & Tessier, 2000). This scale primarily assesses an individual's cognitions related to SCDs, sleep-related daytime dysfunction, and any negative cognition related to sleep and aging. Although not measured directly or asked explicitly, this scale also assesses if an individual may believe their sleep to be a problem. Nevertheless, this scale might be more useful if it also measured an individual's SCDs and compared these SCD with the endorsed cognitions.

The Pittsburg Sleep Quality Index. The PSQI gathers information regarding sleep-related daytime-dysfunction as well as general SCD information (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI is a short questionnaire created to measure an individual's subjective sleep quality for the past month. The PSQI consists of 10 questions, including the participant's bedtime, wake time, overall sleep quality, and sleep environment (Buysse et al., 1989). This measure was validated initially for good sleepers, poor sleepers, and poor sleepers with MDD between ages 24 and 83 years (Buysse et al., 1989), but has since been validated in medical populations, such as individuals with cancer (Beck, A. L. Schwartz, Towsley, Dudley, & Barsevick, 2004). This measure assesses the frequency, chronicity, and severity of SCDs; other sleep

disorders symptoms, such as difficulty breathing; and sleep-related daytime dysfunction. Unfortunately, this measure is also an example assuming these variables are problematic, as there are no questions explicitly asking participants if they consider their SCDs to be problems.

The Insomnia Severity Index. The ISI is a widely-accepted, brief, self-report screening measure for assessing insomnia symptoms. This measure laid the foundations for the questions in the present study and is therefore highly relevant. The measure consists of seven questions scored on a 5-point Likert-scale of 0 through 4. The answers for the seven items are summed, resulting in a score that displays the subjective severity of insomnia symptoms. Higher scores indicate more severe subjective insomnia (Morin, 1993). Bastien, Vallières, and Morin (2001) validated the measure in two separate psychometric studies, the first of which evaluated 145 adults between the ages of 17 and 82. The results of the first study demonstrated that the ISI had an internal consistency of .74 (Bastien, Vallières, & Morin, 2001).

The authors emphasized that there are two parts of the measure: four sleep-related items and three wake-related items (Bastien et al., 2001). Of note, question 1 has three sub-questions that assess the severity of insomnia symptoms by asking respondents to rate their difficulties with falling asleep, staying asleep or waking up too early on a scale of 0 (none) to 4 (very; Bastien et al., 2001; Morin, 1993). The remaining four questions within the ISI assess the respondent's satisfaction or dissatisfaction; how the SCDs interfere with daily functioning; how noticeable the respondent believes their poor sleep is to others; and how worried the individual is regarding their current sleep pattern (Bastien et al., 2001; Morin, 1993). These final four questions are further explored in the

present study, using specific questions to measure sleep-related daytime dysfunction, including feelings of daytime sleepiness or fatigue, difficulty with attention or concentration, and if the participant believes these symptoms of daytime dysfunction are related to their difficulties sleeping. The present study further evaluated these sleep concerns by asking if the participants consider these issues problematic, and these questions comprise the problem endorsement variable—a component that is not covered in the ISI (Morin, 1993; M. L. Perlis, personal communication, May 2018).

Sleep Disorders Symptom Checklist-25. The SCD-CL-25 is a newer, brief assessment tool designed to screen for sleep disorder symptoms in a primary care setting (Klingman, Jungquist, & Perlis, 2017). The measure assesses 25 symptoms of various sleep disorders, including insomnia, OSA, shift work sleep disorder, insufficient sleep disorder, CRSD, RLS, narcolepsy, and parasomnias. For each symptom, individuals can answer on a 4-point Likert scale: never (0), once a month (1), 1-3 times a week (2), 3-5 times a week (3), and more than 5 times a week (4). Once answered, the individual obtains a total overall score between 0 and 125, with sub-scores that can inform the clinician if an individual should be tested for a specific disorder (Klingman et al., 2017). For example, items 10 through 14 assess various symptoms of OSA. If an individual endorses that all five items occur more than five times per week, the clinician may want to consider ordering a polysomnographic sleep study to verify these endorsements (Klingman et al., 2017).

The measure also takes into account demographic and general sleep information, including age, sex, BMI (height and weight), work shifts, work hours, bed partner, typical total sleep time, and typical time in bed. The questionnaire advises individuals to answer

based on the sleep they have experienced over the past 3 months (Klingman et al., 2017). This measure screens primarily for sleep disorder symptoms, as such, and also assesses general SCDs and sleep-related daytime dysfunction. Because this questionnaire was created for use within primary care settings, it may be beneficial for clinicians to follow up with questions of whether the endorsed symptoms can be validated medically.

Advantages and disadvantages of self-report measures. Each of the questionnaires presented comes with its own set of strengths and limitations related to the overarching concerns of assessing for SCD, sleep-related daytime dysfunction, and problem endorsement. Generally, self-report sleep questionnaires have some broader concerns. According to Ibáñez et al. (2018), there can be fluctuations in reporting, causing inconsistencies in how the individual may view their sleep. In addition, the results of the questionnaire may not reflect the true nature of the individual's SCD, sleep-related daytime dysfunction, or sleep-related cognitions. This could be due to lack of candor and social desirability, poor insight into the nature of sleep concerns, misinterpretation of the questions and items, limited options for answering each item, poor or skewed memory of sleep, and a general lack of self-awareness. Finally, there are limitations in the population of individuals completing these questionnaires in clinical and research settings. Specifically, as in all research, individuals presenting to treatment or research studies with sleep concerns may present with voluntary response bias and may not be representative of the general population (Ibáñez et al., 2018).

Despite these limitations, there are many benefits to self-report measures, including that they are easily accessible and are inexpensive in comparison to objective measures, such as PSG. Furthermore, validated subjective self-report measures are an

efficient and cost-effective aid in the diagnosis of sleep disorders, provide clinicians with a structured template for additional questions to ask during intake interviews, and inform clinical decisions (Ibáñez et al., 2018). For example, subjective measures can inform clinicians' decisions related to driving concerns (as with the ESS) or with sleep disorder diagnostic concerns (as with the SDS-CL-25). Finally, subjective measures can provide another perspective to an objective measure. For example, it may be beneficial for an individual to complete a PSQI or SDS-CL-25 in addition to undergoing an in-laboratory polysomnographic sleep study. Ultimately, a multimodal method of measurement may be the best approach to capturing a full picture of patients' sleep and how they subjectively perceive their sleep (Ibáñez et al., 2018).

Objective measures. Whereas subjective/self-report measures rely heavily on individuals' perceptions of their sleep patterns and concerns, objective measures evaluate sleep behavior more directly. Objective measures, such as PSG, use methods including EMG, EOG, and EEG to measure sleep physiology. These measures do not require input from the patient; however, physicians may rely heavily on patient's self-report assessment measures when deciding whether to order an in-laboratory sleep study (Ibáñez et al., 2018).

Polysomnography. PSG is a diagnostic procedure that normally requires an individual to stay overnight at a sleep center. There are at-home options, such as with home-based PSG, which have been shown to be more cost-effective and may be adequate for OSA diagnosis in individuals with high suspicion of the disorder. Nevertheless, the at-home studies are less complicated in their measures. As such, when possible,

clinicians prefer in-laboratory PSG to rule out other sleep disorders that can impact the architecture of sleep (Bruyneel et al., 2011).

The purpose of PSG is to objectively measure aspects of sleep using the following measures: EMG to measure muscle activity, EOG to measure eye movements, ECG to measure heart rhythm, and EEG to measure brain waves. Complete PSG also measures respiratory activity to detect disorders of breathing during sleep (respiratory effort, apneas, and hypopneas; Somers et al., 2008) and measures oxygen saturation levels by use of oximetry (W. S. Chung, Chen & Hang, 2018). The combination of these readings creates a comprehensive profile of sleep behavior and allows for clinicians to rule out almost every *DSM-5* and *ICD-3* sleep disorder (Freedman & Roehrs, 2004).

There is controversy regarding the use of PSG in the diagnosis of insomnia disorder, as it is an objective measure of sleep architecture that does not account for the subjectivity of sleep quality and many disturbances. Moreover, although once common practice, PSG is now infrequently ordered for uncomplicated insomnia disorder alone, mainly due to the cost and validity of other means of assessment (Cunnington, Junge, & Fernando, 2013). Today, this in-lab tool is more often used to diagnose other sleep disorders, such as OSA, REM behavior disorder, PLMD, and other parasomnias, such as nightmares, sleepwalking, and sleep-talking that occur while an individual is asleep (Somers et al., 2008). Often, an individual's subjective sleep quality is not assessed during this evaluation process. Therefore, the PSQI and other questionnaires may also be used in conjunction with PSG to achieve a clearer diagnosis and understanding of presenting problems (Ibáñez et al., 2018).

The present study solely utilizes self-report and does not use objective measures of sleep. This is because the data are archival and consist of individuals who submitted online screening surveys in hopes of participating in studies at the University of Pennsylvania. Although some of these studies have used PSG, the present study did not due to the nature of the dataset. There is, however, a question on the survey that inquires about past PSG studies and can be found within Table 21.

Medical Comorbidities

Research indicates significant associations between sleep duration and multiple medical conditions, such as diabetes, obesity, cardiovascular disease, dementia, and overall mortality (Grandner, 2012). Furthermore, the existence of a sleep disorder, such as insomnia disorder or OSA, can increase the risk of medical comorbidity and mortality (Grandner, Jackson, Pak, & Gehrman, 2012). Regardless of individual demographics, socioeconomic status, and health risk factors, SCDs significantly increase an individual's risk for obesity and associated complications, diabetes, myocardial infarction, stroke, and coronary artery disease (Grandner, Jackson, et al., 2012). It is important to note that demographics and socioeconomic status can impact sleep quality, sleep quantity, and number of sleep disturbances (Grandner et al., 2010).

Cardiovascular disease. Insomnia and SCDs can lower an individual's threshold for developing many diseases, including cardiovascular disease (CVD). Globally, CVD is considered the leading cause of death. These deaths are primarily the result of heart attacks, otherwise known as acute myocardial infarctions. A myocardial infarction occurs when there is tissue death in the heart muscle cells (necrosis) due to a lack of blood flow. This lack of blood flow can occur due to a blockage of the small arteries and

veins that supply the heart with blood and oxygen. The longer the blockage exists, the more likely the heart tissue dies (Hunt, 2005). Like many other acute medical conditions, delayed treatment results in great cell death—time lost is heart lost.

After 20 to 40 minutes, the damage to the heart is irreversible, the affected area of the heart becomes a zone of necrosis (dead tissue), and the heart cells lost can never be recovered (De Luca, Suryapranata, Ottervanger, & Antman, 2004). Individuals experiencing a heart attack may experience chest pain that can extend to the left arm or jaw, sweating, nausea, fatigue, and dyspnea (Greenlund et al., 2004). There are other symptom clusters, which is beyond the scope of this work; however, regardless of presentation, these symptoms are usually a direct cause of the heart muscle not receiving enough oxygen or the SNS response of the body trying to address this physical stressor (Hunt, 2005).

To solidify this relationship between insomnia and CVD, Sofi, Cesari, Casini, Macchi, Abbate, and Gensini (2014) reviewed 13 prospective studies, which included 122,501 participants, in a meta-analysis. The researchers in these 13 individual studies similarly defined insomnia as difficulty falling asleep, difficulty staying asleep, and restless, disturbed sleep. The 13 studies all used self-report methods to assess for insomnia. Sofi et al. observed that the researchers of the 13 studies all followed their participants for lengths of time between 3 and 20 years after baseline. On average, these studies showed that individuals with insomnia are at a 45% or higher increased risk of developing and potentially dying from CVD (Sofi et al., 2014).

Osteoarthritis/chronic pain. Osteoarthritis is a disease involving the degeneration and inflammation of the joints/cartilage, specifically the synovial joint, and,

eventually, bone. Joints are made up of the synovium and articular cartilage that create the inner lining of the joint space between two bones (Felson et al., 2000). One main characteristic of osteoarthritis is the progressive loss of the articular cartilage, which can lead to significant friction between the two bones, causing inflammation and subsequent pain (Felson et al., 2000). Furthermore, the individual may experience sharp aches and burning sensations that become worse with activity, thus making an active lifestyle challenging to maintain (Blagojevic, Jinks, Jeffery, & Jordan, 2010). Of note, the most significant risk factor for osteoarthritis is aging, since the cartilage degrades over an extended period. Thus, the pain caused by this disease is likely to mostly impact adults (30 years or older) rather than younger adults, adolescents, or children (Felson et al., 2000).

Patients suffering from chronic pain, including pain arising from osteoarthritis, can experience SCDs at bedtime, during the night, and upon waking for the day. At bedtime, patients with chronic pain often experience prolonged sleep latency, anxiety, rumination, significant levels of fatigue, and more intense pain (Vitiello et al., 2014). Patients may also experience nightmares, periodic leg movements, apnea, sweating, and heart palpitations (Vitiello et al., 2014), as well as wakefulness during sleep due to pain. According to PSG, patients experiencing pain may spend longer durations of sleep time in stages 1 and 2 and less time in SWS, indicating lighter sleep. Ultimately, this interferes with restorative sleep, negatively impacts daily functioning, and can lower overall sleep quality (Edwards et al., 2009).

Type 2 diabetes. Type 2 diabetes occurs when insulin (a hormone that is produced and released by the pancreas in response to high blood glucose) no longer

works correctly or as effectively. As a result, the body experiences difficulty transporting the glucose from the blood into the cells for energy. This subsequently leads to issues with hyperglycemia, which can also lead to other health complications, such as neuropathy, blindness, hypertension, dementia, stroke, limb damage, and amputation (Turchin et al., 2009). A number of risk factors are responsible for individuals developing diabetes, such as poor diet, sedentary lifestyle, increased BMI, elevated liver-enzyme levels, tobacco use, decreased insulin secretion, and family history (Lyssenko et al., 2008).

Another risk factor for diabetes is insomnia. Vgontzas, Liao, Pejovic, Calhoun, Karatarak, and Bixler, (2009) assessed the relationship between insomnia and type 2 diabetes among 1,741 men and women. The researchers collected health information regarding the participants' health, risk factors for diabetes/diagnosis of diabetes, subjective sleep data, and objective sleep data in the form of in-laboratory PSG (Vgontzas et al., 2009). Regarding sleep data collection, the researchers appeared to measure both sleep-related daytime dysfunction and severity of SCD. They categorized patients as having insomnia if they subjectively reported experiencing insomnia symptoms for 1 year or more. They separated the insomnia group from people whom they categorized as poor sleepers without insomnia. These poor sleepers reported moderate to severe difficulty initiating and maintaining sleep, waking up earlier than intended, or experiencing unrefreshing sleep. Additionally, poor sleepers did not state explicitly that they experienced insomnia. Conversely, individuals within the insomnia group reported four or more symptoms of poor sleep, including explicitly endorsing insomnia. Furthermore, they reported that these symptoms lasted for at least 1 year

(Vgontzas et al., 2009). The researchers also found a significant relationship between chronic insomnia and type 2 diabetes. When compared to individuals who had no insomnia, no sleep-related daytime dysfunction, or slept for more than 6 hours, the individuals with chronic insomnia and sleep durations of 6 hours or less were at a 300% higher risk for developing type 2 diabetes (Vgontzas et al., 2009).

Obesity. Obesity is one of the strongest risk factors for OSA. In individuals with a BMI higher than 40, the prevalence of the disorder can be as high as 90% (A. R. Schwartz et al., 2008). As many as 60% of the individuals referred to PSG for suspected OSA over a 15-year periods had excess body weight as the common clinical characteristic (Punjabi, 2008).

Obesity leads to anatomic alterations in the human body, such as an increase in neck size, waist circumference, and the amount of fat deposited around the upper airway (Somers et al., 2008). These alterations increase the risk of an individual to develop upper airway obstruction during sleep. Also, obesity can lead to a decrease in lung volume, increasing the continuous positive airway pressure requirements (G. D. Foster, Makris, & Bailer, 2005). This can lead to respiratory control system instability and puts greater stress on the lungs and the heart to breathe and to transport oxygen to the brain during sleep (Punjabi, 2008; A. R. Schwartz et al., 2008). Increases in body weight over time not only leads to the onset of OSA and associated health issues, but it can also worsen an OSAHS diagnosis as weight gain continues (Punjabi, 2008).

As indicated, there is a clear relationship between sleep duration and quality, obesity, and metabolic health. Grandner, Schopfer, Sands-Lincoln, Jackson, and Malhotra (2015) determined that this relationship varied across age groups. The

researchers used data from the 2007-2008 National Health and Nutrition Examination Survey (NHANES; N = 5,607) and analyzed respondents' age, sleep duration through self-report, and BMI. As with the present study, age was analyzed continuously and categorically (Grandner et al., 2015). The researchers measured sleep disturbance severity by collecting quantitative TST data and categorized the participants as very short (≤ 4 hours), short (5 to 6 hours), average (7 to 8 hours), or long (≥ 9 hours) sleepers.

The relationship between BMI and sleep duration varied across age groups. For example, in young adults, longer sleep duration was associated with lowered BMI. Conversely, middle-aged adults with the lowest BMI slept 7 to 8 hours each night on average. Long (>9 hours) and short (5 to 6 hours) sleepers during middle age had higher BMIs. The researchers did not specify if these findings were in the early middle age group (30 to 49 years) or the late middle age group (50 to 64 years). Overall, the researchers found a curvilinear relationship between BMI and age. Older adults (65 years or higher) did not appear to show a relationship between sleep and BMI. As such, the researchers postulated that the effects of sleep on BMI (if applicable) have already manifested at this age (Grandner et al., 2015). The researchers focused primarily on sleep duration, categorizing this study as a SCD study. They did not evaluate how sleep length was affected daytime function, if sleep was refreshing, and if the older participants perceived their sleep patterns to be a problem (Grandner et al., 2015).

Stroke. An ischemic stroke occurs when there is a blockage (e.g., a blood clot) in a blood vessel to the brain, substantially reducing the transport of oxygen to the brain tissue. As a result, the tissue that is no longer receiving oxygen begins to die. If the individual is treated within a few hours of onset, he or she is likely to make a better

recovery. After a stroke, a person's life is much affected by the prolonged recovery process, and most individuals have permanent neurologic deficits after a stroke, although to varying degrees; some of these symptoms can improve or recover over time (Sacco et al., 2006).

Although there are many risk factors for stroke, such as hypertension and tobacco use, sleep also appears to play a role. Elwood, Hack, Pickering, Hughes, and Gallacher (2006) utilized data from a cohort study that evaluated lifestyle factors that were considered predictive of vascular disease among 1,986 men between the ages of 55 and 69. The original research specifically assessed if these men were predisposed to a stroke or cardiac event by collecting data on their health, including their sleep. The researchers primarily measured daytime dysfunction with a central focus on daytime sleepiness. SCD, however, was grouped into the same category as having a sleep disorder, such as insomnia, OSA, RLS, or disordered breathing (Elwood, Hack, Pickering, Hughes, and Gallacher, 2006).

The researchers found that approximately one third of these men endorsed at least one symptom of SCD and one third endorsed daytime sleepiness on the Wisconsin Sleep Questionnaire. The results indicated that the risk of a stroke was significantly increased in the men who endorsed at least one symptom of sleep disturbance. This relationship was not significant for the men who only endorsed daytime sleepiness (Elwood et al., 2006). Without specific knowledge of sleep-related daytime dysfunction or measures of SCD severity, it is difficult to determine the accurate strength of this relationship between SCD, sleep-related daytime dysfunction, and stroke.

Neurological Comorbidities

Traumatic brain injury/concussion. A concussion or a mild traumatic brain injury (TBI) usually begins with a blow to the head resulting in a diffuse brain injury. As such, a large area of the brain is affected by the physical impact (N. J. Brown et al., 2014). Depending on the severity of the impact and number of insults, there may be no apparent brain trauma that can be seen on various methods of gross neuroimaging, such as X-ray or MRI. If the injury is more severe, the individual may experience cognitive deficits and behavioral changes (Head, 1993).

Some of these deficits and changes can become chronic. Following a TBI, it is ubiquitous for individuals to experience sleep-wake disturbances, namely insomnia, increased sleep need, and increased daytime sleepiness. The onset of these symptoms can affect the individual's recovery following a TBI (Ouellet, Beaulieu-Bonneau & Morin, 2015). To analyze the prevalence of these sleep-wake disturbances following a TBI, Mathias and Alvaro (2012) performed a meta-analysis using data from 21 studies. The researchers found that approximately 50% of individuals reported experiencing sleep-wake disturbances following TBI and 29% received a clinical diagnosis of insomnia disorder following TBI (Mathias & Alvaro, 2012).

Regarding categorization, this meta-analysis appears to consider studies that evaluate sleep-related daytime dysfunction, as well as SCD severity. Although this was not explicit, the 21 studies included articles that examined the overall prevalence of SCDs in any form following TBI. It also mentioned studies that looked at individuals with newly diagnosed sleep disorders following TBI and studies in which patients exhibited sleep difficulties with subjective and objective measures (Mathias & Alvaro, 2012).

Epilepsy and seizures. Epilepsy is a brain disorder that involves recurring and unpredictable seizures (Lüders, Najm, Nair, Widdess-Walsh, & Bingham, 2006). These epileptic seizures are defined as a “transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain” (Fisher et al., 2005, p. 471). During a seizure, the brain exhibits repeated sudden excitatory signals, which must show alteration in brain activity and be captured on an EEG for a formal diagnosis of epilepsy (Lüders et al., 2006; S. J. M. Smith, 2005). Although all of this abnormal activity is occurring within the brain, externally, the individual may exhibit physical symptoms that are highly dependent on seizure type and location in the brain (Lüders et al., 2006). Seizures can affect sensory, motor, and autonomic functions; consciousness; emotional state; memory; cognition; and behavior (Fisher et al., 2005).

As compared to neurotypical individuals, sleep-wake disturbances are twice as frequent in individuals with epilepsy and evidence suggests that lack of sleep can lower the threshold for seizures in individuals who are predisposed, making these events even more likely (Macêdo, de Oliveira, Foldvary-Schaefer, & da Mota Gomes, 2017). Quigg et al. (2016) evaluated how insomnia disorder is related to the ability to control seizures and how this may, in turn, impact overall quality of life. These researchers collected ISI data for 207 patients with diagnosed epilepsy. Although both portions of the ISI were used, the researchers presented the ISI data as one score and compared the overall scores of individuals with and without seizures (Quigg et al., 2016). As the factors were not separated to measure sleep disturbance severity and daytime dysfunction, this article is difficult to categorize.

Quigg et al. (2016) found that ISI scores were inversely correlated with seizures, in that individuals who experienced epileptic seizures reported higher levels of insomnia severity (ISI scores) compared with those who did not experience seizures. Regarding sleep measures, the researchers included additional data related to SCD severity, such as time in bed, and data for sleep-related daytime dysfunction, such as daytime sleepiness, operationalized as scores on the ESS. Furthermore, the researchers found that worse ESS scores correlated with insomnia (Quigg et al., 2016).

Neurocognitive disorders. Major and mild neurocognitive disorders, formerly known as “Dementia, Delirium, Amnestic, and Other Cognitive Disorders” in the *DSM-IV*, involve the decline of cognitive domains, such as complex attention, executive functioning, language, perceptual-motor abilities, learning and memory, and social cognition (APA, 2013). These symptoms progress into a mild to severe neurocognitive disorder, such as Alzheimer’s disease (AD) or Lewy Body Dementia (LBD; APA, 2013). Unfortunately, with the changes in the brain that occur with these disorders, it is not uncommon for patients with AD or LBD to experience SCD or sleep-related daytime dysfunction (Hennawy, Sabovich, Liu, Herrmann, & Lanctôt, 2019; Osorio et al., 2011).

Alzheimer’s disease. AD is a neurodegenerative disorder that is progressive, incurable, and the most common cause of dementia in elderly individuals. This disease occurs when there is a neuronal loss in the brain, leading to damage, early on, especially in the cortex. Progression of this disease is associated with beta-amyloid plaques and phosphor-tau neurofibrillary tangles accumulate in the brain (Mohandas, Rajmohan, & Raghunath, 2009).

According to the amyloid cascade theory, as the amyloid plaques build up between neurons, there is a disruption in neuron-to-neuron signaling, causing brain function to decline. This can lead to inflammation, which can further damage the brain. The buildup of the beta-amyloid and resultant cell death can lead to the development of neurofibrillary tangles inside and outside of neurons, which can cause apoptosis (cell death), creating a vicious cycle. As a result, the brain begins to atrophy, the gyri become shallower, and the ventricles become larger (Mohandas et al., 2009).

AD is characterized primarily by a progressive decline in memory and areas of cognitive functioning. Initially, symptoms of the disease appear undetectable, but as the plaques and tangles build, individuals begin to develop short-term memory loss, progressing to the loss of motor skills, language production and, eventually, long-term memory loss and disorientation. Late stage AD results in the individual becoming bedridden with death likely the result of infection, such as pneumonia (Dubois et al., 2007).

Lewy body dementia. Patients with LBD must meet criteria for either major or mild neurocognitive disorder (APA, 2013). To be diagnosed with LBD, individuals must exhibit two of the following features: “fluctuating cognition with pronounced variations in attention and alertness, . . . recurrent visual hallucinations that are well formed and detailed, . . . [or] . . . spontaneous features of Parkinsonism with onset subsequent to the development of cognitive decline” (APA, 2013, p. 618). Individuals may also have a diagnosis of REM behavior disorder and or severe neuroleptic sensitivity (APA, 2013).

Sleep Changes with AD and LBD. Individuals with LBD experience SCDs and sleep-related daytime dysfunction in the following ways: excessive daytime napping,

difficulties initiating sleep, frequent nighttime awakenings, and early morning awakenings. Although these SCDs and sleep-related daytime dysfunction also affect the general population, it is of particular consequence in individuals in LBD (Osorio et al., 2011). Bliwise, Mercaldo, Avidan, Boeve, Greer, and Kukull (2011) studied the effects of SCDs on daytime functioning for individuals with LBD as compared to AD. The researchers found that patients with LBD are more likely to experience nighttime SCDs than individuals with AD. Regarding the impact of sleep on disease progression, the researchers also found that individuals with AD who experience nighttime SCDs are also more likely to show a greater progression in the disease than individuals with LBD (Bliwise et al., 2011).

Limited sleep data were collected in the form of a single-item measure asking whether the participant experiences NWAK, EMA, or excessive daytime napping. If any of these symptoms were endorsed, the participant was categorized as having nocturnal sleep disturbances (Bliwise et al., 2011). As such, this study is challenging to categorize given the limited measures and grouping of SCD severity and sleep-related daytime dysfunction into one question. It is also difficult to determine which sub-item drove this relationship and whether it was due primarily to sleep-related daytime dysfunction or to SCDs.

Psychological Comorbidities

Major depressive disorder. MDD is characterized by depressed mood, anhedonia, weight loss or gain, difficulty sleeping or hypersomnia, lethargy, fatigue, feelings of worthlessness, difficulty with concentration or attention, and suicidality (APA, 2013). Two relevant symptom criteria include insomnia or hypersomnia nearly

every day and fatigue or loss of energy nearly every day. Individuals must experience five symptoms for at least 2 weeks to meet diagnostic criteria for MDD. Additional symptoms include feelings of intense sadness, and rumination, all of which can be specifically related to a loss but go beyond “normal” grieving by meeting diagnostic criteria. These significant losses can precipitate depression and can include loss of a loved one, financial hardships, serious medical diagnosis, or loss of belongings due to a natural disaster (APA, 2013). Such triggers are not necessary for a diagnosis of MDD (APA, 2013).

Sleep concerns are a prominent symptom of MDD and research has shown a clear bidirectional relationship between MDD and insomnia disorder (Siversten et al., 2012). In their study on age and sleep disturbances among American men and women, Grandner, Martin, et al. (2012) assessed for depressed mood in addition to SCDs and tiredness. The researchers found that self-report of depressed mood was a significant factor in self-reported SCDs and tiredness. Specifically, a mild depressed mood for fewer than 3 days per week was doubled the likelihood of reporting SCDs and tiredness when compared to individuals with no depressed mood. As the severity of depressed mood increased, so too did the likelihood of reporting SCDs or tiredness (Grandner, Martin, et al., 2012).

Generalized anxiety disorder. Existing literature has also shown a clear relationship between insomnia and anxiety (Ohayon & Roth, 2003). Although this is true for all forms of anxiety, generalized anxiety disorder (GAD) is the most prevalent form of anxiety disorder in older adults, followed by specific phobias (Wolitzky-Taylor, Castriotta, Lenze, Stanley & Craske, 2010). According to the *DSM-5*, GAD affects up to

2.9% of United States adults every year and is defined by the presence of “excessive anxiety and worry” (APA, 2013, p. 223). Of note, anxiety and fear differ in that fear is “the emotional response to real or perceived imminent threat” and results in the activation of the autonomic nervous system in preparation for fight or flight (APA, 2013, p. 189). Conversely, anxiety is the “anticipation of future threat” and is generally characterized by heightened arousal and muscle tension (APA, 2013, p. 189). Many of these anticipations relate to everyday concerns, such as money or family (APA, 2013); however, in the case of maladaptive anxiety, the thoughts related to these stressors become excessive, persistent, and intrusive. Individuals with anxiety disorders may be aware of their disorders but simultaneously believe that the worries are out of their control or that worrying is adaptive, in that it prepares them to deal with anticipated consequences. As a result of these concerns, individuals may also feel irritable, on edge, and have difficulties with attention and concentration (Fricchione, 2004). Physically, individuals may experience a change in appetite, nausea, muscle tension, muscle aches, fatigue, and difficulty sleeping (APA, 2013).

As SCDs are a part of the diagnostic criteria or associated features for all of the anxiety disorders, it is unsurprising that research has sought to assess the relationship and prevalence of insomnia disorder in individuals with anxiety. Ohayon and Roth (2003) collected psychiatric disorder (historical and current) and insomnia information from 14,915 participants between the ages of 15 to 100 in the United Kingdom, Germany, Italy, and Portugal. Their purpose was to investigate psychiatric history, including anxiety disorder history and chronic insomnia. The researchers found that anxiety disorder results varied from other psychiatric disorders, in that a large percentage of

individuals with anxiety found that the onset of anxiety coincided with the onset of their insomnia disorders. They also found that severe levels of insomnia affected 44.6% of individuals with dual diagnoses of MDD and anxiety disorders. There was a significant difference between individuals with dual diagnoses and the 33.6% of individuals with single psychiatric disorders or insomnia disorder diagnoses (Ohayon & Roth, 2003).

Bipolar I disorder. Some hallmark symptoms of bipolar I disorder include dramatic shifts in emotions, and mood and energy levels that can vary from extreme lows to extreme highs. These shifts occur over the course of at least 1 week, to many weeks (APA, 2013). During extreme lows, individuals can experience depression similar to, or meeting diagnostic criteria for MDD, during which they may experience hopelessness, lethargy, difficulties concentrating, anhedonia, and changes in appetite or sleep.

Although depression is a common occurrence for those with bipolar I disorder, it is not a requisite for a diagnosis. In order to meet diagnostic criteria for bipolar I disorder, individuals must experience at least one manic episode, which involves significant energy, excessive happiness or irritability, euphoria, and extremely high self-esteem and delusions of grandeur (APA, 2013). Additionally, individuals experiencing mania may also exhibit pressured speech, racing thoughts, feelings of invincibility, impulsivity, and recklessness, and may experience a “decreased need for sleep (e.g., feels rested after only 3 hours of sleep)” (APA, 2013, p. 124). Furthermore, bipolar I disorder is highly comorbid with anxiety disorders, substance abuse disorders, and ADHD, all of which are also heavily comorbid with insomnia (Crump, K. Sundquist, Winkleby & J. Sundquist, 2013; Hirschfeld, 2014).

Due to the sporadic nature of sleep within bipolar I disorder, individuals may not respond to standard insomnia treatment, such as CBT-I (A. G. Harvey et al., 2015). As such, A. G. Harvey et al. (2015) proposed a bipolar-specific modification of CBT-I, CBTI-BP. They compared individuals who underwent the CBTI-BP protocol ($n = 30$) to individuals who only received psychoeducation ($n = 28$). The CBTI-BP protocol included components of standard CBT-I: stimulus control, sleep restriction, establishing a bedtime routine, and cognitive therapy focusing on dysfunctional beliefs about sleep. There was great emphasis on creating regular rhythms between sleep and wake times, implementing regular daytime rhythms, educating about circadian rhythm, and creating a wake-time routine (A. G. Harvey et al., 2015). Alternatively, the psychoeducation group did not receive any plans to facilitate change. Instead, they received information regarding sleep, stress, daily activities, regulation of bipolar disorder, medications, substance use, relaxation breathing, and self-esteem. When comparing the outcomes of the two groups at follow-up, 73.9% of the individuals who underwent the CBTI-BP protocol no longer met diagnostic criteria for insomnia disorder, whereas only 41.7% of the psychoeducation group no longer met diagnostic criteria for insomnia disorder (A. G. Harvey et al., 2015).

Schizophrenia. Schizophrenia is a mental illness that is characterized primarily by disorganized thinking and/or behavior. Individuals with schizophrenia can have positive symptoms, which can include hallucinations (including but not limited to visual or auditory hallucinations), delusions, disorganized speech and behavior, or catatonic behavior. Individuals may also experience negative symptoms, which involve the diminution of normal processes, such as the reduction of emotional reactivity (flat or

restricted affect), anhedonia (lack of pleasure), alogia (poverty of speech or speech content), or avolition (decrease in motivation). Some more subtle cognitive symptoms include deficits in memory, learning, or understanding (APA, 2013; Laurens et al., 2015).

Schizophrenia may also be comorbid with SCDs and sleep-related daytime dysfunction (Kaskie, Graziano, & Ferrarelli, 2017). Individuals with schizophrenia, when in psychotic states, often exhibit episodes of sleep deprivation. This, often unintended, sleep deprivation can exacerbate symptoms of schizophrenia (Wulff, Dijk, Middleton, R. G. Foster, & Joyce, 2012). Schizophrenia patients in acute states of psychosis may experience worsening of hallucinations and delusions, which may be associated with arousal and, therefore, impair sleep (Kaskie et al., 2017). Individuals with schizophrenia primarily experience early or middle insomnia (Haffmans, Hoencamp, Knegtering, & van Heycop Ten Ham, 1994). Others may experience a complete reversal, in which they sleep during the day and are awake at night. Other subjective sleep reports include agitation, feelings of restlessness, distressing hypnagogic hallucinations, and disturbing nightmares (Lee & Douglass, 2010). Despite these complications, insomnia presents at the same rate in schizophrenia patients when compared to healthy controls (Benson, 2015).

Summary of the Literature

Although older adults experience greater difficulty falling and staying asleep, and experience more medical and psychiatric comorbidities than younger individuals, there is emerging evidence that older individuals are less likely to report that difficulty initiating sleep, maintaining sleep, or waking up earlier than intended is problematic (Ohayon et al., 2004). This discordance between SCD, sleep-related daytime dysfunction, and the

perception that these symptoms are problems is unusually prevalent among older adults, making them less likely to seek treatment (Grandner, Martin, et al., 2012). As such, they may be at greater risk for the development (or exacerbation) of multiple medical and psychiatric disorders such as cardiovascular disease, diabetes, obesity, stroke, dementia, anxiety, and depression (Bredesen, 2018; Grandner, Martin, et al., 2012). Despite the plethora of sleep literature, few studies have simultaneously assessed SCD and sleep-related daytime dysfunction, and even fewer have assessed problem endorsement.

CHAPTER 3: METHOD

Research Design and Justification

The retrospective study utilized a cross-sectional group comparison approach to assess for age differences in relation to SCD, sleep-related daytime dysfunction, and percent problem endorsement in an archival/community dataset consisting of individuals with sleep complaints.

Participants

For the original dataset, participants ($N = 4,069$) were between the ages of 18 and 89 ($M_{\text{age}} = 38.23$; $SD = 14.55$), were primarily comprised of women (61.49%), and a majority of the participants were White ($n = 2,567$). The present study consisted of 932 participants between the ages of 18 and 89 ($M_{\text{age}} = 46.49$; $SD = 17.80$). Of these participants, 53.6% were women and a majority of participants were White ($n = 736$).

Inclusion criteria. Archival data were used. Participants who completed initial surveys were eligible if they reported difficulty sleeping. Participants completed questions online regarding demographic data, current sleep patterns, and medical and psychological comorbidities. A stable sleep pattern and a diagnosis for insomnia disorder was not a requirement for participation. Further, participants were required to have (a) been age 18 or older, (b) completed the survey online from May 4, 2011 through October 13 2018, (c) given consent for their submissions to be used as part of research, (d) given authorization for Confidentiality of Records and Health Insurance Portability and Accountability Act (HIPAA) forms, (e) submitted a completed survey, and (f) been located within the greater Philadelphia area at the time of survey completion.

Exclusion criteria. Potential participants were excluded if they (a) were under the age of 18, (b) completed the survey prior to May 4, 2011, (c) did not give consent for their submissions to be used as part of research, (d) did not authorize Confidentiality of Records or HIPAA forms, (e) submitted a partially completed survey, and (e) were not located within the greater Philadelphia area at the time of survey completion.

Participants were not excluded from the dataset for any comorbid sleep disorder, medical condition, psychological condition, or substance use.

Recruitment. The original data for this study were collected through an online website at www.sleeplessinphilly.com, a recruitment site established and utilized by the Behavioral Sleep Medicine Program at the University of Pennsylvania. Participants were directed to this website through several sources: (a) online advertisements, (b) television advertisements, (c) radio advertisements, (d) paper brochures, (e) referrals from physicians, (e) referrals from friends, or (f) direct e-mail through the university's electronic medical records system.

Measure: Sleepless in Philly

The Sleepless in Philly online questionnaire is based on an earlier online tool used at the University of Rochester called Sleepless in Rochester. This tool was created by Dr. Michael L. Perlis during his professorship at the University of Rochester. Upon Perlis's transfer to the University of Pennsylvania, he adapted this tool for the University of Pennsylvania's Behavioral Sleep Medicine Program, of which he is Director. The www.sleeplessinphilly.com survey was launched in 2009 and continues to be utilized in 2019 as a recruitment tool for the studies conducted at the University of Pennsylvania's Behavioral Sleep Medicine Program (M.L. Perlis, personal communication, July 6,

2019). There is no formal publication of this survey and this survey was included with permission from its aforementioned creator. Permission was granted for inclusion in this document on July 6, 2019 and letter of permission is available upon request. Of note, the www.sleeplessinphilly.com survey has been updated several times since its original launch date. The versions between May 4, 2011 and October 2018 are the most consistent. As such, versions that existed prior to May 4, 2011 were eliminated from the dataset for the present study.

The www.sleeplessinphilly.com online questionnaire consists of six sections and 103 questions, with an additional five female-specific questions. The six sections are as follows: (a) General Information, (b) Basic Sleep/Insomnia Information, (c) Sleep-Related Questions, which assessed for other sleep disorders (d) General Health Questions, which assessed for medical and psychiatric comorbidities (e) Chronic Pain Question, and (f) Treatment for Insomnia/Sleeplessness, which evaluated whether individuals previously sought treatment for insomnia or sleep disturbances. The present study primarily utilized the General Information and Basic Sleep/Insomnia Information sections.

Participants completed 28 questions in the General Information section (Section 1), including age, gender, race and ethnicity, education level, occupation, and typical hours at work, and 37 questions in the Basic Sleep/Insomnia Information section, categorized into the three main variables (SCD, Sleep-Related Daytime Dysfunction, and Problem Endorsement), each encompassing an important aspect of sleep (Section 2 of 6). The questions within this section encompassed the severity, frequency, and chronicity of respondents' SCD; whether they experienced any sleep-related daytime dysfunction; and

whether the SCDs reported were perceived to be problematic. The item location and breakdown for each variable can be found below. Please refer to Appendix B for the complete www.sleeplessinphilly.com survey.

SCD. SCD encompasses difficulty falling asleep (initial insomnia), staying asleep (middle insomnia), waking up a number of times throughout the night, and waking up earlier than desired (late insomnia) as assessed by self-report on the online survey, www.sleeplessinphilly.com. These disturbances were assessed in the collected data regarding SL, NWAK, WASO, and EMA. Participants were asked to quantify their typical sleep durations or TSTs in hours. SCD questions relevant to the present study can be found in Items 33, 37, 41, 46, and 50 within “Section 2 of 6: Basic Sleep/Insomnia Information” on the www.sleeplessinphilly.com survey (See Appendix B).

Sleep-related daytime dysfunction. Sleep-related daytime dysfunctions are symptoms related to SCD. These symptoms were assessed using the www.sleeplessinphilly.com survey, and included symptoms of attention or concentration difficulties, feeling rested upon waking, impaired daytime function, fatigue, and sleepiness. As such, a total score of sleep-related daytime dysfunction ranged from 0 to 5. Specifically, participants who reported no sleep-related daytime dysfunction received a score of 0 and participants endorsing all five of the sleep-related daytime dysfunction symptoms yielded a score of 5. Of note, the question, “Do you feel rested upon waking?” was reverse scored. Finally, sleep-related daytime dysfunctions were evaluated independently to determine specific age-related differences. Sleep-related daytime dysfunction questions relevant to the present study can be found in Items 55, 58, 59, 60,

61 within “Section 2 of 6: Basic Sleep/Insomnia Information” on the www.sleeplessinphilly.com survey (See Appendix B).

Problem endorsement. Problem endorsement was assessed by a specific question, “Do you consider this a problem?” As such, participants had an opportunity to endorse this if each of their SCDs was considered problematic. Problem endorsement questions were evaluated independently to determine specific age-related differences for each SCD. Questions regarding problem endorsement can be found on Items 35, 39, 43, 48, and 52 within “Section 2 of 6: Basic Sleep/Insomnia Information” on the www.sleeplessinphilly.com survey (See Appendix B). Of note, these items followed questions related to SCD, so that they specifically asked participants if they considered their SL, NWAK, WASO, EMA, and TST to be problems.

Demographics. Participant were required to provide demographic information on Items 1 through 28 on “Section 1 of 6: General Information” within the www.sleeplessinphilly.com survey. The demographic information that was particularly relevant to the present study included sex (Item 16), race (Item 21), height (Item 18), and weight (Item 19). The question of sex required the individual to choose from one of two options: male or female. Unfortunately, the survey did not allow for additional options. For race, individuals were asked with which of the following race options they most closely identified: American Indian or Alaska Native, Asian, Black or African American, Native Hawaiian or Other Pacific Islander, or White. Individuals were also asked to provide their heights in feet/inches and their weights in pounds. The participants’ height and weight responses were used to automatically compute for BMI. From this number, the individuals were categorized as underweight ($< 18.5 \text{ kg/m}^2$), normal weight (18.5 to

24.9 kg/m²), overweight (25 to 29.9 kg/m²), or obese (≥ 30 kg/m²). In addition, participants were asked to provide their dates of birth when completing “Section 1 of 6: General Information.” This single question can be found in Item 17 of the General Information section on the www.sleeplessinphilly.com survey (See Appendix B).

Procedure

Prior to the present study, participants between the ages of 18 and 89 were completed an online questionnaire at www.sleeplessinphilly.com. Participants provided consent and granted permission to retain information for future use in research. Individual participant data were collected at one time point and the dataset for the present study was collected over the course of 9 years. Since its conception in 2009, the survey has been revised eight times. The latter three versions were utilized in this study’s data as they are the most similar in nature, collecting the same demographic and necessary sleep data in a similar fashion. As such, the screening forms from May 4, 2011 through October 13, 2018 were utilized.

Participants completed demographic information, including education level, occupation, and general work hours. Participants also answered questions related to basic sleep and insomnia information, additional sleep disorders, and physical and mental health. Participants were categorized into one of four age groups: young adult (YA; 18 to 29 years), adult (A; 30 to 44 years); middle age (MA; 45 to 64 years), or older adult (OA; 65 to 89 years). The age group with the least participants was the OA age group. As such, the composition of this age group was evaluated and the YA, A, and MA participants were matched to OAs by sex, race, and BMI. Matching was completed using a random number generator of the participants’ ID numbers. These numbers were

automatically assigned to the participants upon submission of their www.sleeplessinphilly.com surveys.

The dataset was compiled and analyzed by the responsible investigator. This dataset was obtained with permission from Perlis. Use of this online questionnaire was evaluated and approved by the Institutional Review Board of the University of Pennsylvania. The request to use archival data collected through this online questionnaire was approved by the Institutional Review Board of Philadelphia College of Osteopathic Medicine.

Statistical methodology. The program Statistical Package for the Social Sciences (SPSS 24.0) was used to perform statistical analyses to test the aforementioned hypotheses. The alpha level for each test was set at $\alpha = 0.05$. Averages for SL, NWAK, WASO, EMA, and TST were calculated for each age group (YA, A, MA, and OA). Values greater than three standard deviations from the mean of each group were removed. Following the removal of outliers by age group for values greater than three standard deviations for SL, NWAK, WASO, EMA, and TST, the data ($N = 932$) were matched the OA ($n = 233$) group because this was the age group with the smallest number of individuals. The age groups mentioned above were first matched based on the variables of sex, then race, and then BMI. Specifically, individuals from each variable were selected using a random number generator of the participant's ID number—a number given automatically when the participant submitted their www.sleeplessinphilly.com survey. If there was no pairing for individuals of similar BMI, they were paired with the closest number within the BMI category above or below that number. This was specifically the case for several underweight individuals who

were then paired with individuals on the lower end of the normal weight category. The purpose of matching participants was twofold: to create equal groups and to control for the variables of sex, race, and BMI, thus creating fewer potential confounds.

CHAPTER 4: RESULTS

Demographic Data

Once outliers were removed, the sample consisted of 932 participants. These participants were matched based on age, sex, race, and BMI. As such, the age groups were demographically similar in regard to the variables of sex, race, and BMI. Overall, the sample consisted of 53.6% women and 46.4% men (See Table 1).

Table 1

Primary Demographic Data by Age Group – Gender

	<i>n</i>	Gender	
		Male (%)	Female (%)
Young Adults	233	108 (46.4%)	125 (53.6%)
Adults	233	108 (46.4%)	125 (53.6%)
Middle Age	233	108 (46.4%)	125 (53.6%)
Older Adults	233	108 (46.4%)	125 (53.6%)
Total	932	432 (46.4%)	500 (53.6%)

Note: Groups were matched by gender, BMI, and race.

The sample consisted mostly of White participants (79.7%), followed by Black or African American participants (19.5%). There were very few individuals of Asian (0.4%) and American Indian/Alaska Native (0.4%) background (See Table 2).

Table 2

Primary Demographic Data by Age Group – Race

	<i>n</i>	Race			
		American Indian or Alaska Native (%)	Asian (%)	Black or African American (%)	White (%)
Young Adults	233	1 (0.4%)	1 (0.4%)	45 (19.5%)	184 (79.7%)
Adults	233	1 (0.4%)	1 (0.4%)	45 (19.5%)	184 (79.7%)
Middle Age	233	1 (0.4%)	1 (0.4%)	45 (19.5%)	184 (79.7%)
Older Adults	233	1 (0.4%)	1 (0.4%)	45 (19.5%)	184 (79.7%)
Total	932	4 (0.4%)	4 (0.4%)	180 (19.5%)	736 (79.7%)

Note: Groups were matched by gender, BMI, and race.

Regarding BMI, most of the individuals fell within the overweight category (25 to 29.9 kg/m²; 40.8%), followed by obese (≥ 30 kg/m²; 31.3%), and normal weight (18.5 to 24.9 kg/m²; 26.9%). Underweight individuals (< 18.5 kg/m²) made up the smallest percent of the sample (1.0%; See Table 3).

Table 3

Primary Demographic Data by Age Group – BMI

	<i>n</i>	BMI			
		Underweight ^a (%)	Normal Weight ^b (%)	Overweight ^c (%)	Obese ^d (%)
Young Adults	233	3 (1.3%)	62 (26.6%)	95 (40.8%)	73 (31.3%)
Adults	233	1 (0.4%)	64 (27.5%)	95 (40.8%)	73 (31.3%)
Middle Age	233	2 (0.9%)	63 (27.0%)	95 (40.8%)	73 (31.3%)
Older Adults	233	3 (1.3%)	62 (26.6%)	95 (40.8%)	73 (31.3%)
Total	932	9 (1.0%)	251(26.9%)	380 (40.8%)	292 (31.3%)

Note: Groups were matched by gender, BMI, and race.

^aUnderweight was categorized as less than 18.5 kg/m²

^bNormal weight was categorized as between 18.5 and 24.9 kg/m²

^cOverweight was categorized as between 25 and 29.9 kg/m²

^dObese was categorized as greater than 30 kg/m²

Recruitment data. In regard to recruitment, most individuals obtained information for the original study (where this data was acquired) through Craigslist advertisements published by the University of Pennsylvania's Behavioral Sleep Medicine Program (33.3%), followed by other methods (e.g., Google search, family friend, or clinical research search; 16.0%) and the University of Pennsylvania website (11.4%; See Table 4).

Table 4

Frequency of Recruitment Methods for Sample

	Frequency	Percent
Bala Clinic	2	0.2%
Craigslist Ad	310	33.3%
Flyer	52	5.6%
Friend	88	9.4%
Health Care Provider	54	5.8%
Health Fair	1	0.1%
Internet Ad	24	2.6%
Internet Search Engine	50	5.4%
Letter from PCP	15	1.6%
Mall Poster	3	0.3%
Metro	32	3.4%
Other	149	16.0%
Radio	17	1.8%
Text Message	7	0.8%
Transit Shelter	1	0.1%
TV Advertisement	6	0.6%
UPenn Almanac	15	1.6%
UPenn Website	106	11.4%

Hypothesis I

A MANOVA was conducted to determine age group differences on SCD variables, including SL (minutes), NWAK (frequency), WASO (minutes), EMA (minutes), and TST (minutes). TST was converted from hours to minutes for continuity. To verify this analysis, a series of five one-way ANOVAs were conducted. To reduce

the probability of a type I error, a Bonferonni correction was utilized and the alpha level was adjusted at $\alpha = .001$.

For each MANOVA and one-way ANOVA, Tukey and Games-Howell post hoc analyses were utilized to determine specific between-groups differences. Effect sizes (Cohen's d) were calculated to determine the magnitude of change between the minimum and maximum values and between the youngest and oldest groups (YA and OA). Error bars on the graphs represent one standard deviation from the mean for each group.

MANOVA results for age and SCD. A MANOVA determined that there were significant differences between age groups for SCD in general, Wilk's $\Lambda = .884$, $F(15,2145.36) = 6.54$, $p < .001$ (See Tables 5).

The Levene's test was not found to be statistically significant for SL ($p = .327$) or NWAK ($p = .056$); therefore, equal variances for these variables can be assumed across groups. Conversely, the Levene's test was found to be statistically significant for WASO ($p < .001$), EMA ($p < .001$), and TST ($p = .014$); therefore, equal variances for these variables cannot be assumed across groups. The test of between-subjects effects was examined and no significant difference were found between age groups for average sleep latency ($p = .095$). Significant differences were found between age groups for NWAK ($p = .008$), WASO ($p < .001$), EMA ($p < .001$), and TST ($p < .001$).

Table 5

MANOVA Summary Table for Age Group and Sleep Continuity Disturbances

Dependent Variable	df	df error	F	Age Group	Means	95% Confidence Interval	
						Lower Bound	Upper Bound
SL (minutes)	3	2145.36	2.13	YA	48.72	43.94	53.49
				A	46.14	41.32	50.97
				MA	44.55	40.04	49.07
				OA	40.43	35.83	45.02
NWAK (frequency)	3	2145.36	3.98	YA	2.57	2.31	2.83
				A	2.76	2.49	3.03
				MA	3.19	2.94	3.44
				OA	2.83	2.58	3.09
WASO (minutes)	3	2145.36	11.14	YA	27.25	21.84	32.65
				A	36.86	31.41	42.32
				MA	44.40	39.29	49.51
				OA	47.51	42.31	52.71
EMA (minutes)	3	2145.36	9.99	YA	53.70	47.95	59.44
				A	56.69	50.88	62.49
				MA	68.79	63.36	74.23
				OA	72.33	66.80	77.85
TST (minutes)	3	2145.36	8.24	YA	365.11	353.51	376.71
				A	357.39	345.67	369.12
				MA	329.71	318.74	340.69
				OA	337.54	326.37	348.70

Games-Howell post hoc analyses were utilized to further assess differences in age group for NWAK, WASO, EMA and TST. For NWAK, significant differences were found between the YA ($M = 2.57$, $SD = 1.79$) and MA ($M = 3.19$, $SD = 2.21$) groups ($p = .011$). For WASO, significant differences were found between the YA ($M = 27.25$, $SD = 29.59$) and A ($M = 36.86$, $SD = 36.91$) groups ($p = .030$), between the YA and MA ($M = 44.40$, $SD = 44.64$) groups ($p < .001$), and between the YA and OA ($M = 47.51$, $SD = 37.36$) groups ($p < .001$). There were also significant differences between the A and OA groups ($p = .026$). For EMA, YAs ($M = 53.70$, $SD = 34.58$) were significantly different from MAs ($M = 68.79$, $SD = 49.52$; $p = .002$) and OAs ($M = 72.33$, $SD = 35.85$; $p < .001$); and As ($M = 56.69$, $SD = 37.83$) were significantly different from MAs ($p = .032$) and OAs ($p < .001$). Finally, for TST, YAs ($M = 365.11$, $SD = 94.58$) were significantly

different from MAs ($M = 329.71$, $SD = 78.58$; $p < .001$) and OAs ($M = 337.54$, $SD = 76.40$; $p = .009$); and As ($M = 357.39$, $SD = 73.37$) were significantly different from MAs ($p = .002$) and OAs ($p = .047$). Table 6 illustrates these results.

Table 6

MANOVA Means and Standard Deviations for Sleep Continuity Disturbances by Age Group

	Age Group	<i>n</i>	Mean	Standard Deviation
SL	YA	188	48.72	35.21
	A	184	46.14	33.38
	MA	210	44.55	34.79
	OA	203	40.43	29.84
	Total	785	44.86	33.42
NWAK	YA	188	2.57	1.79
	A	184	2.76	1.50
	MA	210	3.19	2.21
	OA	203	2.83	1.76
	Total	785	2.85	1.86
WASO	YA	188	27.25	29.59
	A	184	36.86	36.91
	MA	210	44.40	44.64
	OA	203	47.51	37.36
	Total	785	39.33	38.45
EMA	YA	188	53.70	34.58
	A	184	56.69	37.83
	MA	210	68.79	49.52
	OA	203	72.33	35.85
	Total	785	63.25	40.81
TST	YA	188	365.11	94.58
	A	184	357.39	73.37
	MA	210	329.71	78.58
	OA	203	337.54	76.40
	Total	785	365.11	82.14

One-way ANOVAs for age and SCD. A series of one-way ANOVAs were conducted to evaluate whether the age group differences for SCDs would withstand Bonferonni corrections. Following the Bonferonni correction, the alpha level for each test was set at 0.05. The purpose of the one-way ANOVAs was to further evaluate group differences for the SCD of SL, NWAK, WASO, EMA, and TST (See Tables 7 and 8).

Table 7

One-Way ANOVAs' Means and Standard Deviations for Sleep Continuity Disturbances by Age Group

	Age Group	<i>n</i>	Mean	Standard Deviation
SL	YA	233	46.88	36.09
	A	233	43.64	33.94
	MA	233	43.87	35.13
	OA	233	39.73	30.59
	Total	932	43.53	34.04
NWAK	YA	233	2.34	1.77
	A	233	2.56	1.58
	MA	233	3.10	2.18
	OA	233	2.71	1.71
	Total	932	2.68	1.84
WASO	YA	233	24.61	28.43
	A	233	33.50	35.66
	MA	233	44.41	46.57
	OA	233	44.71	36.99
	Total	932	36.80	38.34
EMA	YA	188	53.70	34.58
	A	184	56.68	37.83
	MA	210	68.79	49.52
	OA	203	72.33	35.85
	Total	785	63.25	40.81
TST	YA	233	373.39	93.75
	A	233	367.21	81.37
	MA	233	334.76	82.89
	OA	233	349.44	85.74
	Total	932	356.20	87.26

Table 8

Summary Table for Series of One-Way ANOVA Results for Age Group and Sleep Continuity Disturbances

	Symbols ^a	Sig. (<i>p</i>)	Data	Effect Size		
				Groups (Min-Max)	Values (Min-Max)	YA-OA
SL (min)	OA = MA = A = YA	.159	39.73 = 43.87 = 43.64 = 46.88	YA-OA	0.214	0.214
NWAK (freq)	OA = (MA > A = YA)	< .001*	2.71 = (3.10 > 2.56 = 2.34)	YA-MA	-0.383	-0.213
WASO (min)	(OA = MA) > A > YA	< .001*	(44.71 = 44.41) > 33.50 > 24.61	YA-OA	-0.609	-0.609
EMA (min)	(OA = MA) > (A = YA)	< .001*	(72.33 = 68.79) > (56.68 = 53.7)	YA-OA	-0.529	-0.529
TST (min)	OA = (MA < A = YA); OA < YA	< .001*	349.44 = (334.76 < 367.21 = 373.39); 349.44 < 373.39	YA-MA	0.437	0.267

* $p < .001$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and SL. A one-way ANOVA was conducted and revealed no significant difference between age groups in SL in minutes, $F(3,928) = 1.73$, $p = .159$ (See Table 9). The Levene's test was not found to be significant and equal variances can be assumed, $p = .389$. As such, there were no significant differences for SL between YAs ($M = 46.88$; $SD = 36.09$), As ($M = 43.64$; $SD = 33.94$), MAs ($M = 43.87$; $SD = 35.13$), and OAs ($M = 39.73$; $SD = 30.59$; See Graph 1). As such, post hoc analyses were not conducted and there was a very weak effect size between YAs and OAs ($d = 0.214$).

Table 9

One-Way ANOVA Results for Age Group and Sleep Latency in Minutes

SL Minutes	<i>df</i>	Sum of Squares	Mean Square	<i>F</i>	<i>p</i>
Between groups	3	6009	2003	1.73	.159
Within groups	928	1072809	1156		
Total	931	1078818			

* $p < .001$

Graph 1

Sleep Latency (in Minutes) by Age Group



Note: SL Minutes by Age Group: OA = MA = A = YA, $p = .159^a$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and NWAK. A one-way ANOVA was conducted and found significant overall difference between age groups on NWAKs, $F(3,928) = 7.21, p < .001$ (See Table 10). The Levene's test was found to not be significant; thus equal variances can be assumed, $p = .139$.

Table 10

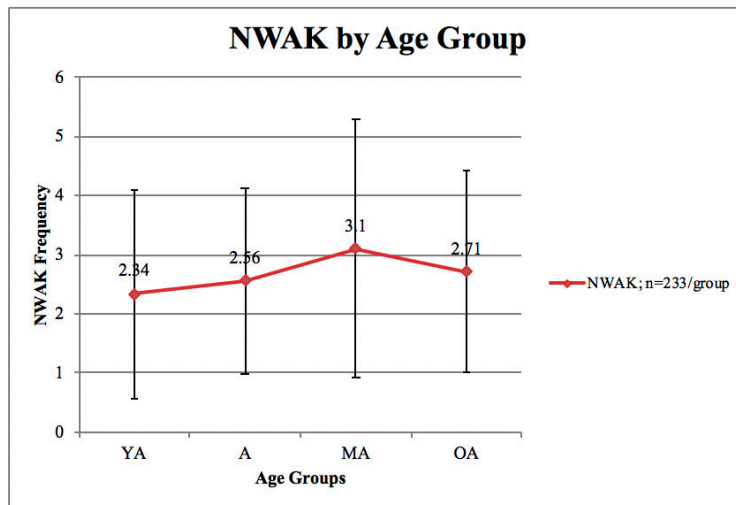
One-Way ANOVA Results for Age Group and Number of Nighttime Awakenings

NWAK Frequency	<i>df</i>	Sum of Squares	Mean Square	<i>F</i>	<i>p</i>
Between groups	3	72	24	7.21	.000*
Within groups	928	3081	3		
Total	931	3153			

* $p < .001$

A Tukey HSD post hoc analyses was conducted and significant differences were found between YAs ($M = 2.34; SD = 1.77$) and MAs ($M = 3.10; SD = 1.71; p < .001; d = -0.383$), as well as between As ($M = 2.56; SD = 1.58$) and MAs ($p = .007$; See Graph 2). No significant differences were found between YAs and As ($p = .582$) or between OAs ($M = 2.71; SD = 1.71$) and YAs ($p = .128; d = -0.213$), As ($p = .797$), or MAs ($p = .096$). Therefore, YAs and As woke up significantly fewer times during the night than MA individuals, but not significantly less times than OAs.

Graph 2

Nighttime Awakenings (Frequency) by Age Group

Note: NWAK Frequency by Age Group: OA = (MA > A = YA), $p < .001^a$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and WASO. A one-way ANOVA was conducted and determined overall significant differences between age groups and WASO in minutes, $F(3, 827.49) = 15.49$, $p < .001$ (See Table 11). The Levene's test was found to be significant so equal variances cannot be assumed, $p < .001$. As such, a Brown-Forsythe was conducted to determine overall significant difference.

Table 11

One-Way ANOVA Results for Age Group Wake After Sleep Onset in Minutes

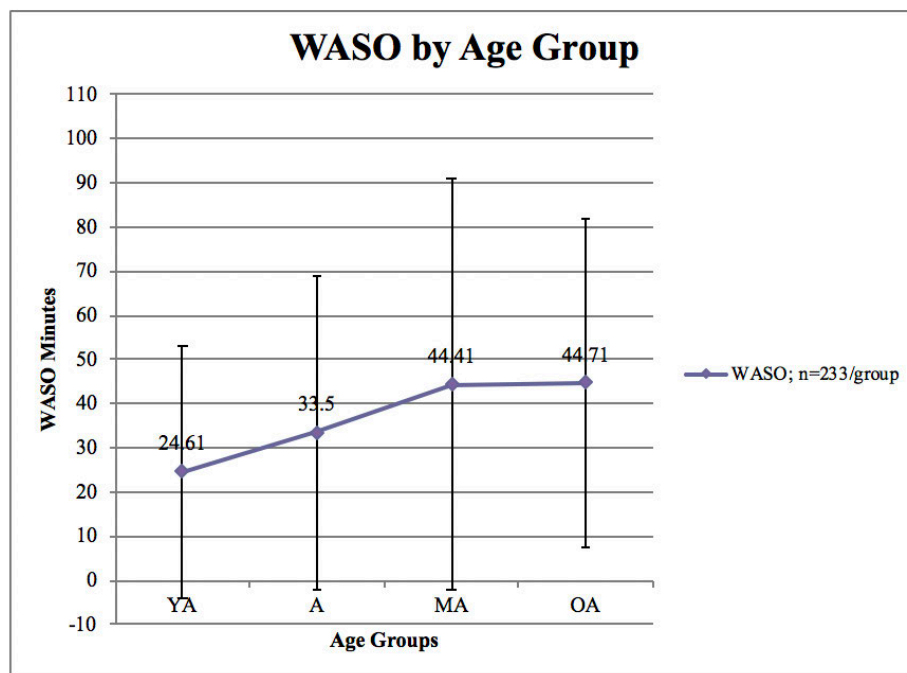
WASO Minutes	<i>df</i>	Sum of Squares	Mean Square	<i>F</i>	<i>p</i>
Between groups	3	65248	21749	15.49	.000*
Within groups	928	1303054	1404		
Total	931	1368302			

* $p < .001$

A Games-Howell post hoc analysis revealed significant differences between YAs ($M = 24.61$; $SD = 28.43$) and As ($M = 33.50$; $SD = 35.66$; $p = .016$), MAs ($M = 44.41$; $SD = 46.57$; $p < .001$), and OAs ($M = 44.71$; $SD = 36.99$; $p < .001$; $d = -0.609$; See Graph 3). YAs were awake for significantly less time during the night than As, MAs, and OAs. Significant differences were also found between As and MAs ($p = .024$) and OAs ($p = .005$). Therefore, A were awake for significantly less time than MAs and OAs. No significant differences were found between MAs and OAs ($p = .999$). This means that MAs and OAs were awake for similar times during the night. An illustration of these findings can be found on Graph 9.

Graph 3

Wake After Sleep Onset (in Minutes) by Age Group



Note: WASO Minutes by Age Group: (OA = MA) > A > YA, $p < .001^a$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and EMA. A one-way ANOVA was conducted and overall significant difference between age groups and EMA in minutes, $F(3, 731.08) = 10.15, p < .001$ (See Table 12). The Levene's test was found to be significant so equal variances cannot be assumed, $p < .001$. As such, a Brown-Forsythe test was conducted and an overall significant difference was found between YAs ($M = 53.70; SD = 34.58$)/As ($M = 56.68; SD = 37.83$) and MAs ($M = 68.79; SD = 49.52$)/OAs ($M = 72.33; SD = 35.85$). Taken together, OAs and MAs woke up significantly earlier than As and YAs (See Table 12).

Table 12

One-Way ANOVA Results for Age Group and Early Morning Awakenings in Minutes

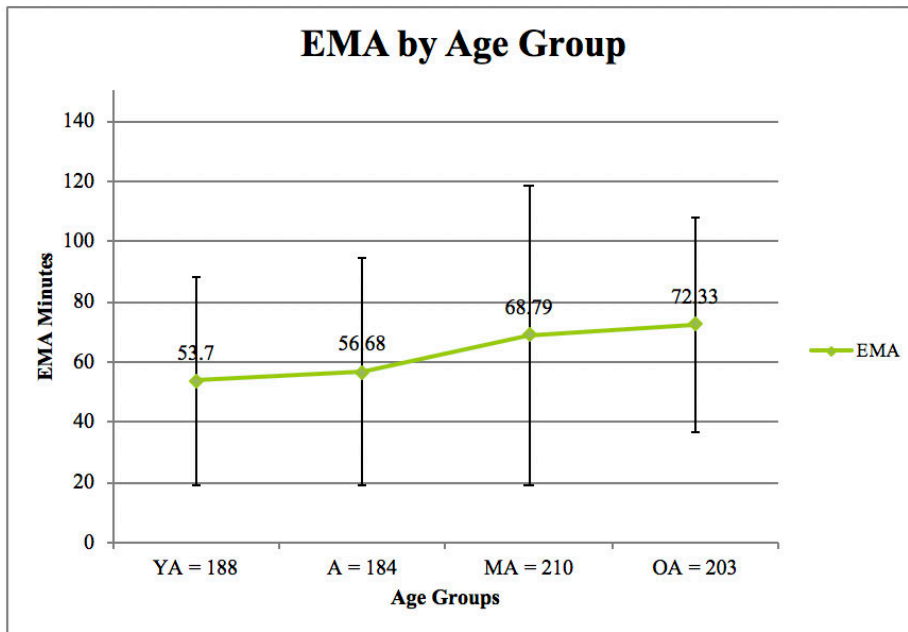
EMA Minutes	<i>df</i>	Sum of Squares	Mean Square	<i>F</i>	<i>p</i>
Between groups	3	48253	16084	9.99	.000*
Within groups	781	1257567	1610		
Total	784	1305820			

* $p < .001$

A Games-Howell post hoc analysis was conducted and revealed significant differences in EMA between YAs and MAs ($p = .002$) and OAs ($p < .001; d = -0.529$), as well as between As and MAs ($p = .032$) and OAs ($p < .001$). No significant differences were found between YAs and As ($p = .857$) or between MAs and OAs ($p = .839$; See Graph 4).

Graph 4

Early Morning Awakenings (in Minutes) by Age Group



Note: EMA Minutes by Age Group: (OA = MA) > (A = YA), $p < .001^a$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and TST. A one-way ANOVA was conducted and determined overall significant difference between age groups and TST in minutes, $F(3,928) = 9.67$, $p < .001$ (See Table 13). The data for TST were originally collected in hours but recalculated into minutes for continuity with the other variables. The Levene's test was found to not be significant; thus equal variances can be assumed, $p = .106$.

Table 13

One-Way ANOVA Results for Age Group and Total Sleep Time in Minutes

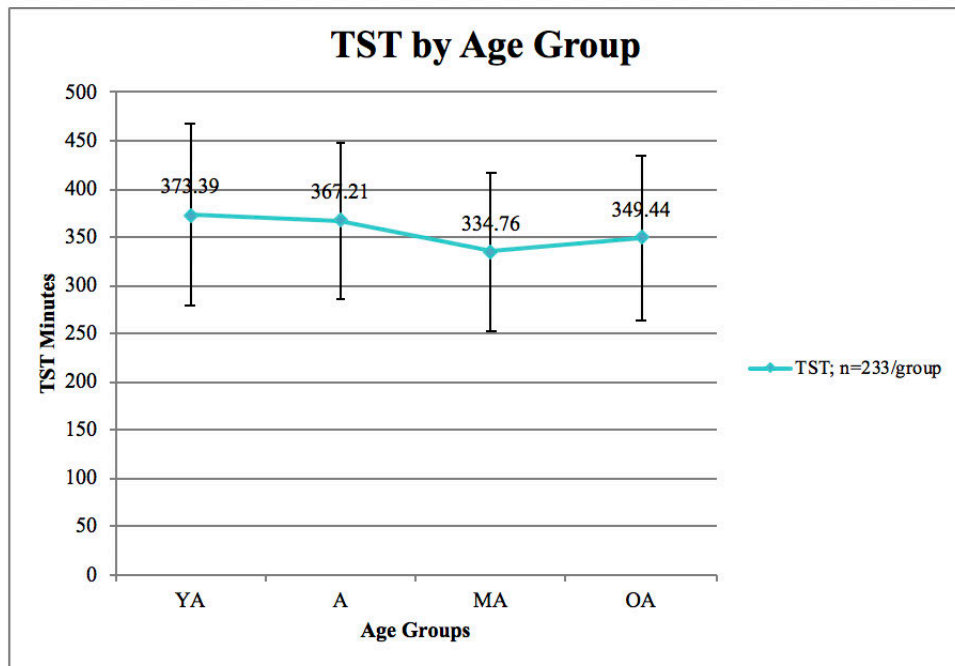
TST Minutes	<i>df</i>	Sum of Squares	Mean Square	<i>F</i>	<i>p</i>
Between groups	3	214806	71602	9.67	.000*
Within groups	928	6874547	7407		
Total	931	7089354			

* $p < .001$

A Tukey HSD post hoc analyses was conducted and significant differences were found between YAs ($M = 373.39$; $SD = 93.75$) and MAs ($M = 334.76$; $SD = 82.89$; $p < .001$; $d = 0.437$), between YAs and OAs ($M = 349.44$; $SD = 85.74$; $p = .015$; $d = 0.267$); as well as between As ($M = 367.21$; $SD = 81.37$) and MAs ($p < .001$). No significant differences were found between As and YAs ($p = .866$), MAs and OAs ($p = .255$), or As and OAs ($p = .116$). Therefore, YAs slept significantly longer than MAs and OAs, and As slept significantly longer than MAs (See Graph 5).

Graph 5

Total Sleep Time (in Minutes) by Age Group



Note: TST Minutes by Age Group: OA = (MA < A = YA); OA < YA, $p < .001^a$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Hypothesis II

A series of five chi-square tests of independence were conducted to assess for relationships between age group and specific sleep-related daytime dysfunction variables, including self-reported concentration, restedness, impaired daytime function, fatigue, and daytime sleepiness. Overall, the five chi-square tests of independence only yielded a significant relationship between age group and concentration, $X^2(3, N = 932) = 18.24, p < .001$ (See Table 14).

A one-way ANOVA was conducted but did not determine age group differences in total sleep-related daytime dysfunction index, $F(3, 928) = 1.86, p = .135$ (See Table

15). For the statistical analyses in Hypothesis II, a Bonferonni correction was utilized and the alpha level was adjusted at $\alpha = .001$.

Table 14

Contingency Analyses for Sleep Related Daytime Dysfunction

	Symbols ^a	Sig. (<i>p</i>)	Data	Cramer's V	Phi Statistic	% Δ		
						Groups (Min- Max)	Values (Min- Max)	YA- OA
Concentration	OA < MA < A < YA	< .001*	57.1% < 67.4% < 71.7% < 74.3%	.140	.020	OA- YA	23.2%	23.2%
Rested (Recoded)	OA = MA = A = YA	.884	76.8% = 79.0% = 77.3% = 79.4%	---	---	OA- YA	3.3%	-3.3%
Impaired Daytime Dysfunction	OA = MA = A = YA	.206	71.7% = 76.4% = 77.3% = 70.0%	---	---	YA-A	10.4%	2.4%
Fatigue	OA = MA = A = YA	.623	84.1% = 83.3% = 83.3% = 79.8%	---	---	YA- OA	5.4%	5.4%
Daytime Sleepiness	OA = MA = A = YA	.400	78.5% = 81.1% = 84.6% = 82.4%	---	---	OA-A	7.8%	-4.7%
Total Average Score	YA=A=MA=OA	.424	3.27=3.39=3.29=3.15	---	---	OA-A	0.207	0.102

* $p < .001$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA). INDICATE SIGNIFICANCE HERE AND TABLE

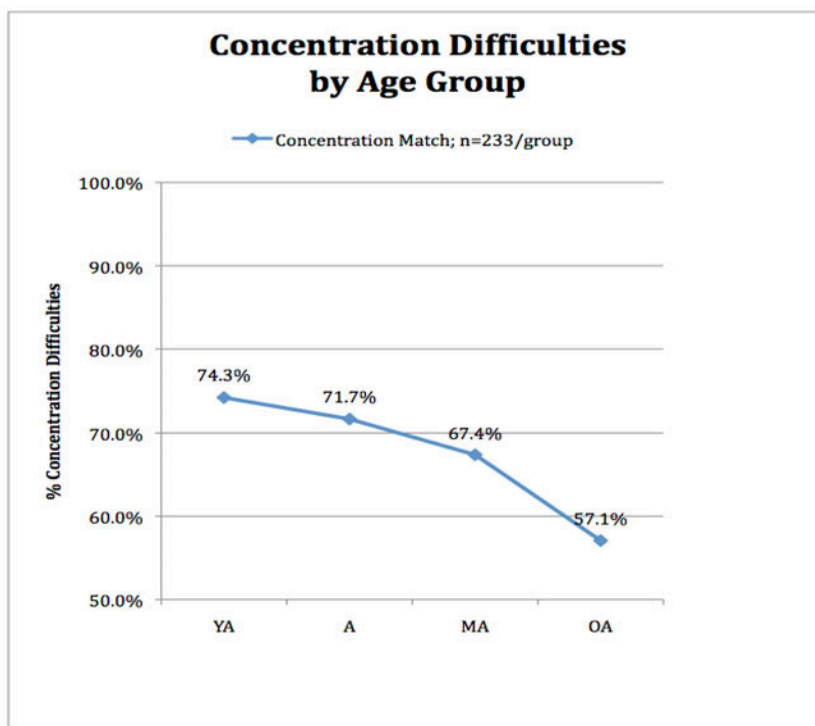
Age and concentration. A chi-square test of independence was conducted and found a relationship between age group and percent of individuals who endorsed concentration difficulties, $\chi^2 (3, N = 932) = 18.24, p < .001$ (See Table 14).

Cramer's V and phi statistic were conducted and, despite significance, the relationship was determined to be weak with a small effect size, $\phi_c = .140, \phi = .020$. The percent changes between YAs and OAs (-23.2%)—which are also the largest and

smallest problem endorsement groups, respectively—reflected an approximate 20% difference. As such, there was a significant but weak relationship between age groups and concentration difficulties (See Graph 6).

Graph 6

Concentration Difficulties by Age Group



Note: Percent Endorsement of Concentration Difficulties by Age Group: $OA < MA < A < YA$, $p < .001^a$

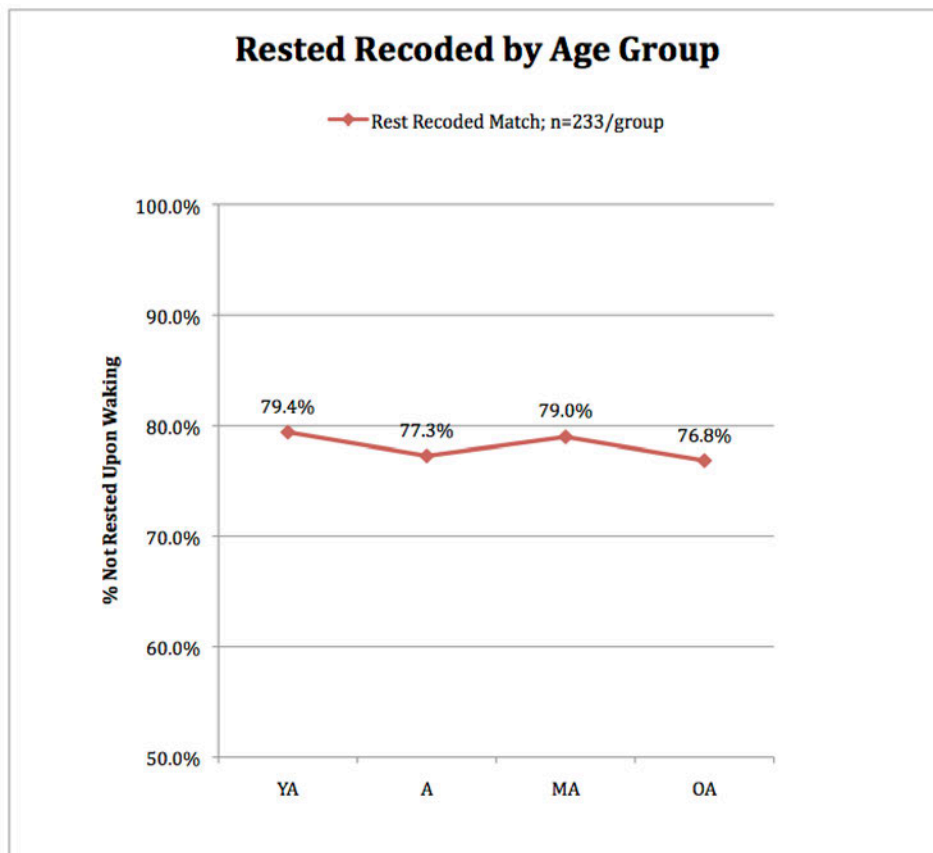
^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and restedness. A chi-square test of independence was conducted and found no significant relationship between age group and percent of individuals who endorsed feeling rested upon waking, $\chi^2 (3, N = 932) = 0.65$, $p = .884$ (See Table 14).

Prior to this analysis, this variable was recoded wherein “yes” responses were given values of 0 and “no” responses were given values of 1 YAs and OAs (-3.3%), which are also the largest and smallest problem endorsement groups, respectively, reflected a very minimal change. In sum, there was no significant relationship between age groups and the endorsement of being rested upon waking (See Graph 7).

Graph 7

Rested (Recoded) by Age Group



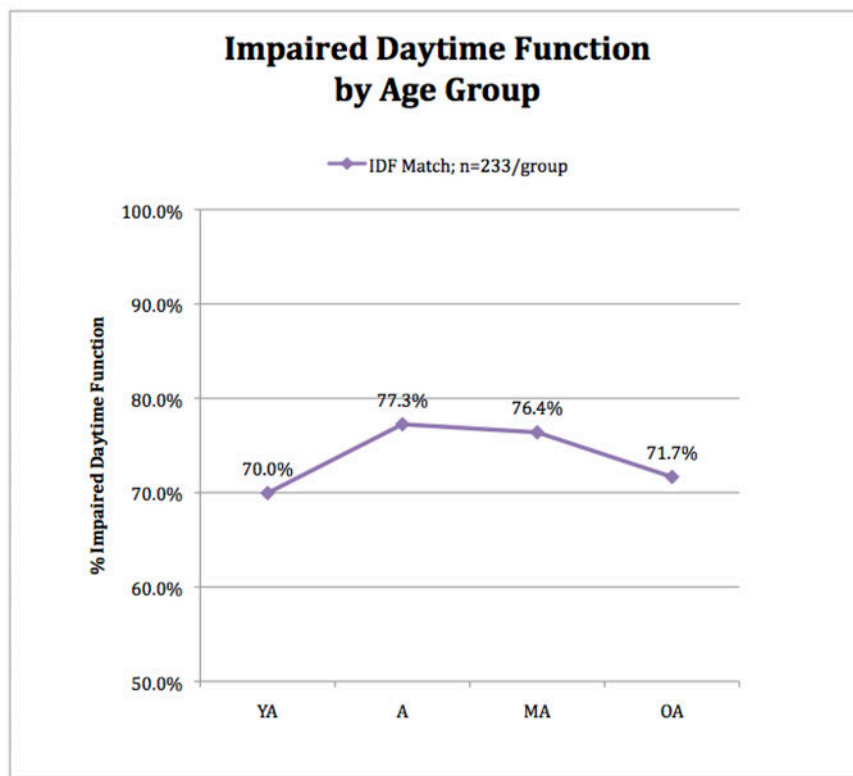
Note: Percent Endorsement of Rested Recoded by Age Group: OA = MA = A = YA, $p = .884^a$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and impaired daytime function. A chi-square test of independence was conducted and found no significant relationship between age group and percent of individuals who endorsed impaired daytime function, $\chi^2 (3, N = 932) = 4.58, p = .206$ (See Table 14). The percent changes between YAs and OAs (2.4%) and between the smallest percent endorsement (YA) and largest percent endorsement (MA; 10.4%) were not statistically significant. As such, there was no significant relationship between age groups and individuals who experienced impaired daytime function (See Graph 8).

Graph 8

Impaired Daytime Function by Age Group



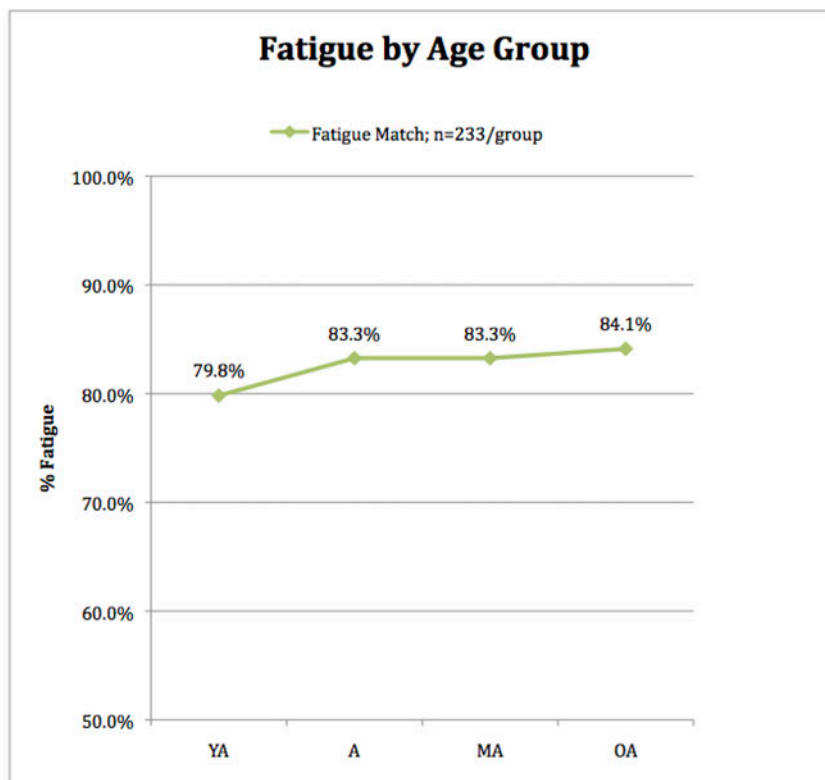
Note: Percent Endorsement of Impaired Daytime Function by Age Group: $OA = MA = A = YA, p = .206^a$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and fatigue. A chi-square test of independence was conducted and determined no significant relationship between age group and percent of individuals who endorsed fatigue during the day, $\chi^2 (3, N = 932) = 1.76, p = .623$ (See Table 14). The percent changes between YAs and OAs (5.4%), which are also the smallest percent endorsement group and largest percent endorsement group, were minimal. As such, there was no significant relationship between age groups and individuals who experienced daytime fatigue (See Graph 9).

Graph 9

Fatigue by Age Group



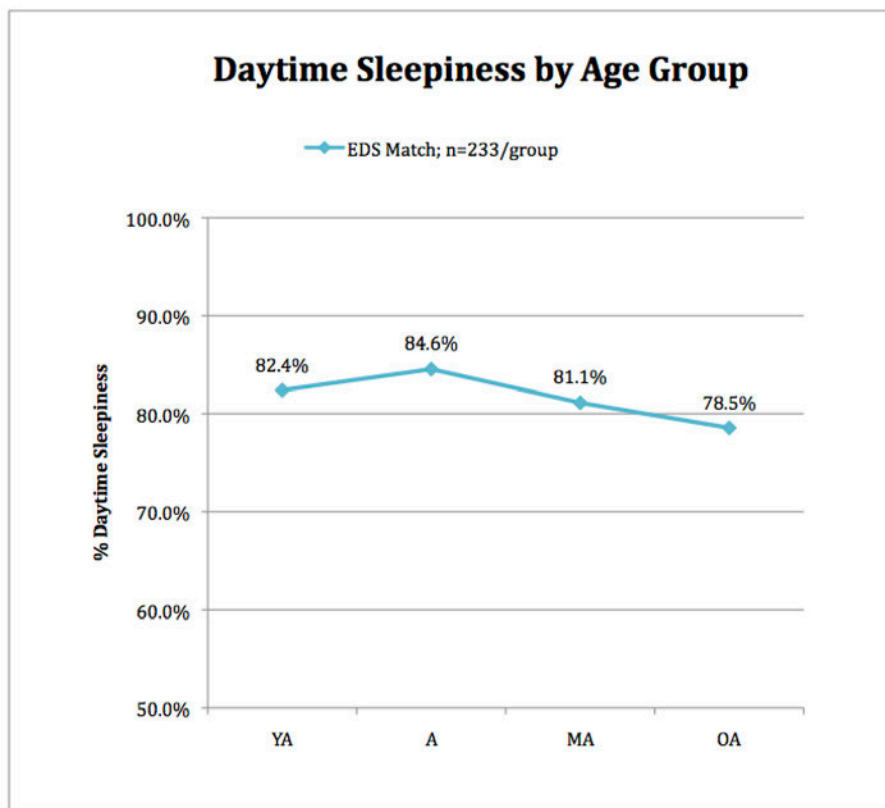
Note: Percent Endorsement of Fatigue by Age Group: OA = MA = A = YA, $p = .623^a$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and daytime sleepiness. A chi-square test of independence was conducted and found no significant relationship between age group and percent of individuals who endorsed daytime sleepiness, $\chi^2 (3, N = 932) = 2.94, p = .400$ (See Table 14). The percent changes between YAs and OAs (-4.7%) and between the smallest percent endorsement (OA) and largest percent endorsement (A; 7.8%) were not statistically significant. As such, there was no significant relationship between age groups and individuals who experienced daytime sleepiness (See Graph 10).

Graph 10

Daytime Sleepiness by Age Group



Note: Percent Endorsement of Daytime Sleepiness by Age Group: OA = MA = A = YA, $p = .400^a$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and total sleep-related daytime dysfunction. A one-way ANOVA was conducted and determined no significant difference between age groups and Sleep-Related Daytime Dysfunction Scaled Score, $F(3, 928) = 1.86, p = .135$ (See Tables 14 and 15). The Sleep-Related Daytime Dysfunction Scaled Score was calculated by applying a value to the “yes” (1) and “no” (0) options for each specific sleep-related daytime dysfunction variable of Concentration Difficulties, Rested (recoded), Impaired Daytime Function, Fatigue, and Daytime Sleepiness. Taken together, each individual was able to obtain a score between 0 and 5, depending on the number of items endorsed. The recoded Rested variable was used in the computation of this new variable to ensure continuity.

Table 15

One-Way ANOVA Results for Age Group and Sleep-Related Daytime Dysfunction Index

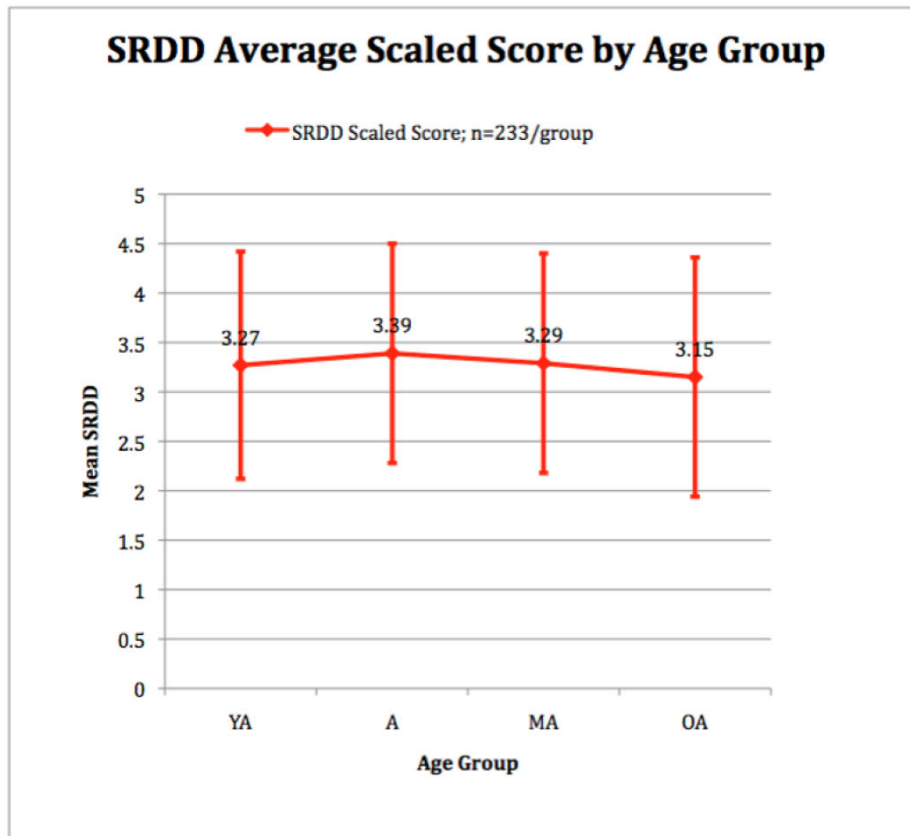
Sleep-Related Daytime Dysfunction Index	<i>df</i>	Sum of Squares	Mean Square	<i>F</i>	<i>p</i>
Between groups	3	7	2.43	1.859	.135
Within groups	928	1215	1.31		
Total	931	1222			

* $p < .001$

The Levene’s test was found to not be significant; thus equal variances can be assumed, ($p = .424$). Overall significance between-groups was not found for YAs ($M = 3.27, SD = 1.15$), As ($M = 3.39, SD = 1.11$), MAs ($M = 3.29, SD = 1.11$), and OAs ($M = 3.15, SD = 1.21$). As such, there were no significant differences between age groups and average of self-reported Sleep-Related Daytime Dysfunction Scaled Score (See Graph 11).

Graph 11

Sleep-Related Daytime Dysfunction Scaled Score by Age Group



Note: Average Sleep-Related Daytime Dysfunction Scaled Score by Age Group: OA = MA = A = YA, $p = .400^a$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Hypothesis III

A series of five chi-square tests of independence were conducted for categorical data to assess the relationships between age group and percent of problem endorsement for each SCD. These tests yielded significant relationships between age groups and problem endorsement for NWAK, WASO, EMA, and TST (See Table 16).

A Bonferonni correction was utilized and the alpha level was adjusted at .001. A phi statistic was calculated to estimate effect size. For significant results, Cramer's V was conducted to determine the strength of each contingency analysis and the relationship between categorical data. Of note, the questionnaire only required individuals with reported SL, NWAK, WASO, or EMA of greater than 0 to answer questions about problem endorsement for each SCD. For all variables, percent changes were calculated to show differences in the minimum and maximum percent problem endorsement and differences in younger and older age groups (YA and OA).

Table 16

Contingency Analyses for Problem Endorsement of Sleep Continuity Disturbances

		Sig. (<i>p</i>)	Data	Cramer's V	Phi Statistic	% Δ		
	Symbols ^a					Groups (Min- Max)	Values (Min- Max)	YA-OA
SL (min)	OA = MA = A = YA	.832	78.4% = 78.1% = 79.0% = 75.5%	.033	.001	YA-A	4.6%	3.8%
NWAK (freq)	MA > OA > A > YA	< .001*	81.3% > 79.0% > 76.7% > 63.4%	.159	.025	YA-MA	28.2%	24.6%
WASO (min)	MA > OA > YA > A	< .001*	93.5% > 88.1% > 84.8% > 74.8%	.190	.036	YA-MA	25.0%	17.8%
EMA (min)	OA > MA > A > YA	< .001*	82.7% > 80.5% > 69.7% > 56.3%	.233	.054	YA-OA	46.9%	46.9%
TST (min)	MA > OA > A > YA	< .001*	78.1% > 76.4% > 66.1% > 57.9%	.178	.032	YA-MA	35.4%	32.0%

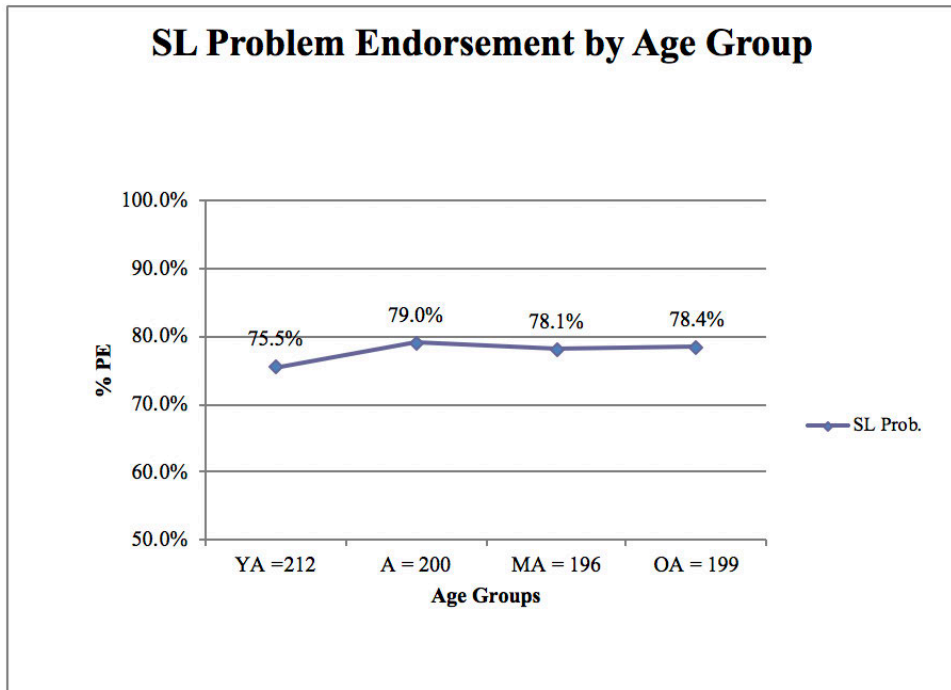
**p* < .001

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and SL problem endorsement. A chi-square test of independence was conducted and found no significant relationship between age group and percent of individuals who endorsed SL as a problem, $\chi^2 (3, N = 807) = 0.87, p = .832$ (See Table 16). Additionally, the percent changes between the YA and OA groups (3.8%) and between the group with the smallest percent endorsement (YA) and the largest percent endorsement (A; 4.6%) were minimal. There was no relationship between age group and problem endorsement for SL and there were no differences in how YAs, As, MAs, and OAs reported similar levels of problem endorsement for SL (See Graph 12).

Graph 12

Sleep Latency Percent Problem Endorsement by Age Group



Note: SL Problem Endorsement by Age Group: $OA = MA = A = YA, p = .832^a$

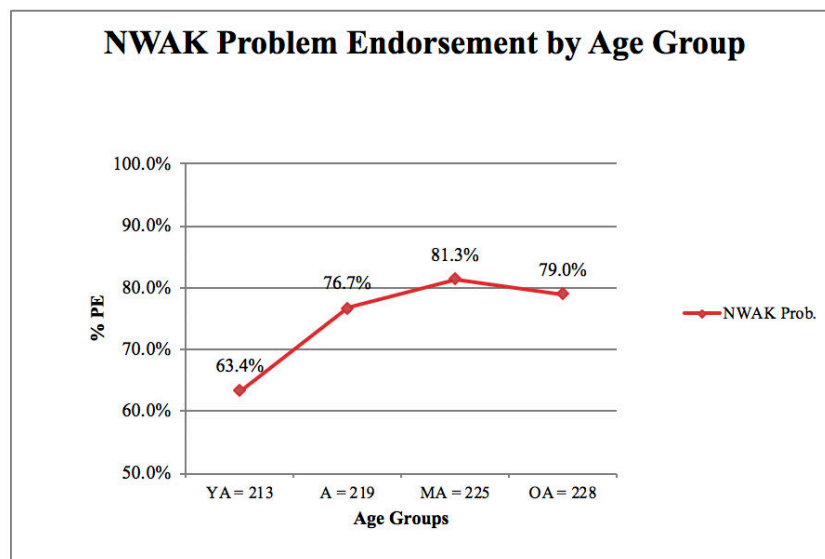
^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and NWAK problem endorsement. A chi-square test of independence was conducted and found a significant relationship between age group and percent of individuals who endorsed NWAK as a problem, $\chi^2 (3, N = 885) = 22.51, p < .001$ (See Table 16).

Cramer's V and phi statistic were conducted and, despite significance, the relationship was determined to be weak with a small effect size, $\phi_c = .159, \phi = .025$. The percent changes between YAs and OAs (24.6%) and between the smallest percent endorsement (YA) and largest percent endorsement (MA; 28.2%) were between 20% and 30%. In sum, there was a significant but weak relationship between YAs, As, MAs, and OAs and their problem endorsement of NWAK (See Graph 13).

Graph 13

Nighttime Awakening Percent Problem Endorsement by Age Group



Note: NWAK Problem Endorsement by Age Group: $MA > OA > A > YA, p < .001^a$

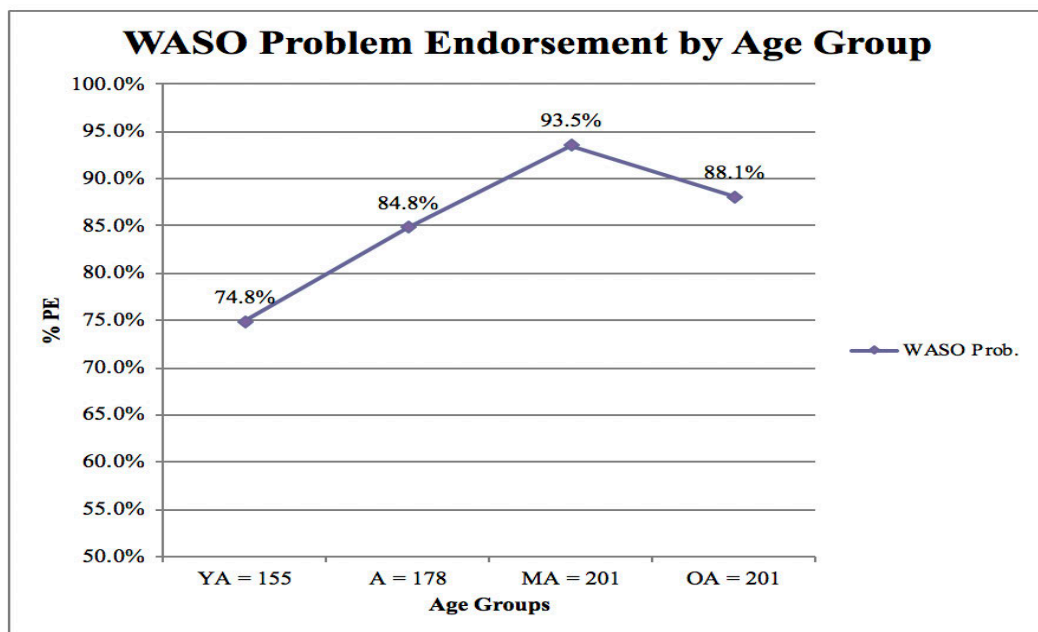
^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and WASO problem endorsement. A chi-square test of independence was conducted and found a relationship between age group and percent of individuals who endorsed WASO as a problem, $\chi^2 (3, N = 735) = 26.40, p < .001$ (See Table 16).

Cramer's V and Phi statistic were conducted and, despite significance, the relationship was determined to be weak with a small effect size, $\phi_c = .190, \phi = .036$. The percent changes between YAs and OAs (17.8%) and between the smallest percent endorsement (YA) and largest percent endorsement (MA; 25.0%) were modest. In sum, there was a significant but modest relationship between age group and problem endorsement of WASO (See Graph 14).

Graph 14

Wake After Sleep Onset Percent Problem Endorsement by Age Group



Note: WASO Problem Endorsement by Age Group: $MA > OA > YA > A, p < .001^a$

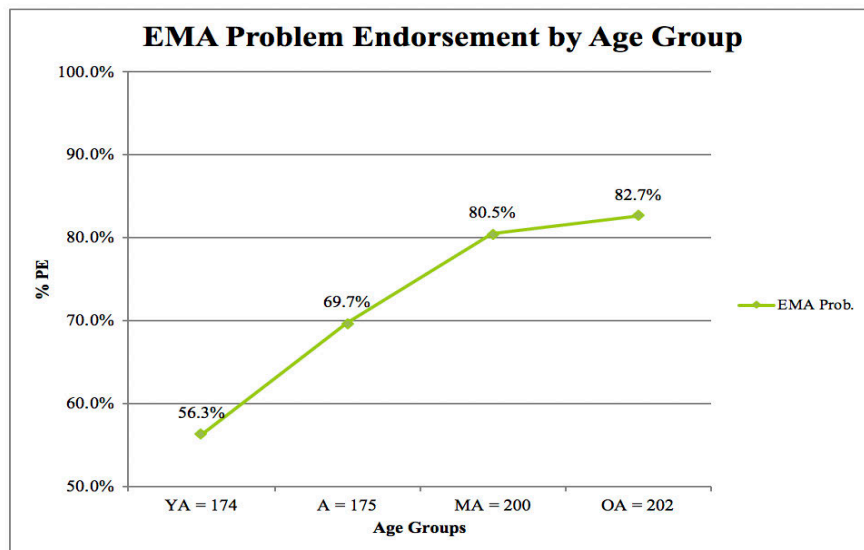
^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and EMA problem endorsement. A chi-square test of independence was conducted and found a significant relationship between age group and percent of individuals who endorsed EMA as a problem, $\chi^2 (3, N = 751) = 40.78, p < .001$ (See Table 16).

Cramer's V and Phi Statistic were conducted and the relationship was determined to be moderate, $\phi_c = .233, \phi = .054$. The percent changes between YAs and OAs (46.9%)—which are also the smallest and largest problem endorsement groups, respectively—reflected almost a 50% difference. As such, there was a significant moderate relationship between age groups and problem endorsement of EMA (See Graph 15).

Graph 15.

Early Morning Awakenings Percent Problem Endorsement by Age Group



Note: EMA Problem Endorsement by Age Group: $OA > MA > A > YA, p < .001^a$

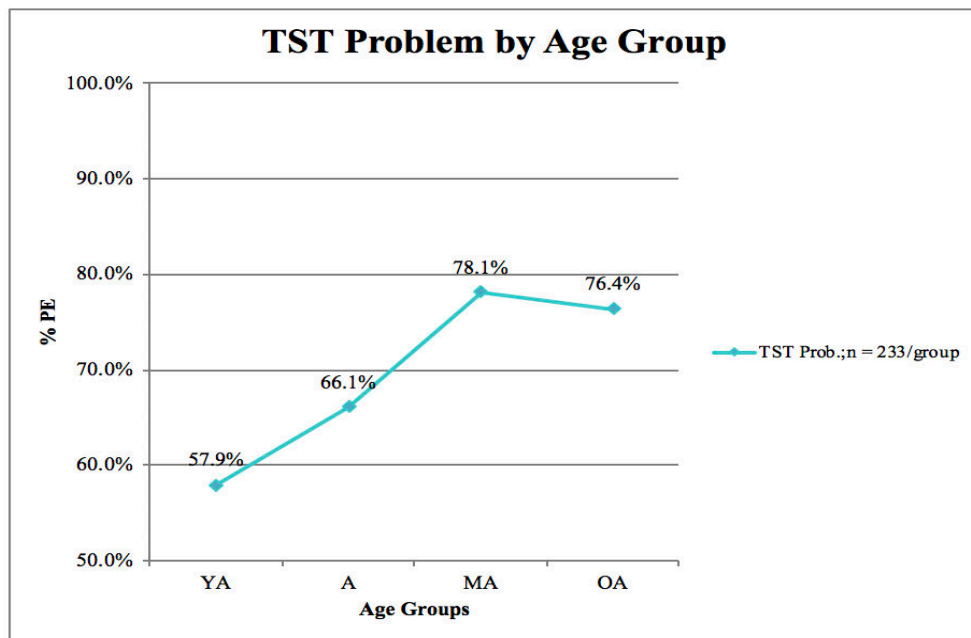
^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Age and TST problem endorsement. A chi-square test of independence was conducted and determined a significant relationship between age group and percent of individuals who endorsed TST as a problem, $\chi^2 (3, N = 932) = 29.41, p < .001$ (See Table 16).

Cramer's V and Phi Statistic were conducted and, despite significance, the relationship was determined to be weak, $\phi_c = .178, \phi = .032$. There were very similar percent changes between YAs and OAs (32.0%) and between YAs and MAs (35.4%). In sum, there was a significant but weak relationship between age groups and problem endorsement of TST (See Graph 16).

Graph 16

Total Sleep Time Percent Problem Endorsement by Age Group



Note: TST Problem Endorsement by Age Group: $MA > OA > A > YA, p < .001^a$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Additional Frequency Analyses

Although not included in the hypotheses, frequency analyses were performed to develop a greater understanding of the current sample. Specifically, frequency analyses were performed for additional demographic data, including ethnicity (See Table 17), education (See Table 18), occupation (See Table 19), sleep disorder symptoms (See table 20), and additional insomnia symptoms (See Table 21), as well as medical and psychiatric comorbidities and related questions (See Tables 22, 23, and 24, respectively). The frequency analyses determined the distribution of these variables across age groups.

Table 17

Secondary Demographic Data by Age Group – Ethnicity

	<i>n</i>	Ethnicity	
		Hispanic	Non-Hispanic
Young Adults	212	26 (12.3%)	186 (87.7%)
Adults	206	13 (6.3%)	193 (93.7%)
Middle Age	195	5 (2.6%)	190 (97.4%)
Older Adults	188	1 (0.5%)	187 (99.5%)
Total	801	45 (5.6%)	756 (94.4%)

Table 18

Secondary Demographic Data by Age Group – Education

	<i>n</i>	Levels of Education							
		No HS ^a	Some HS ^a	HS/GED ^a	Some College	Associate's Degree	Bachelor's Degree	Master's Degree	Doctorate
Young Adults	233	1 (0.4%)	1 (0.4%)	35 (15.0%)	66 (28.3%)	13 (5.6%)	89 (38.2%)	24 (10.3%)	4 (1.7%)
Adults	233	0 (0.0%)	4 (1.7%)	34 (14.6%)	61 (26.2%)	15 (6.4%)	63 (27.0%)	41 (17.6%)	15 (6.4%)
Middle Age	233	3 (1.3%)	5 (2.1%)	51 (21.9%)	55 (23.6%)	16 (6.9%)	50 (21.5%)	38 (16.3%)	15 (6.4%)
Older Adults	233	1 (0.4%)	5 (2.1%)	23 (9.9%)	51 (21.9%)	10 (4.3%)	59 (25.3%)	51 (21.9%)	33 (14.2%)
Total	932	5 (0.5%)	15 (1.6%)	143 (15.3%)	233 (25.0%)	54 (5.8%)	261 (28.0%)	154 (16.5%)	67 (7.2%)

^aHS is the abbreviation used for High School.

Table 19

Secondary Demographic Data by Age Group – Occupation

	<i>n</i>	Occupation								
		Professional/ Technical/ Managerial	Clerical/ Scales	Service	Processing	Structural Work	Misc. ^a	Unemployed	Disabled	Other
Young Adults	233	35 (15.0%)	7 (3.0%)	27 (11.6%)	2 (0.9%)	4 (1.7%)	16 (6.9%)	58 (24.9%)	3 (1.3%)	81 (34.8%)
Adults	233	84 (36.1%)	17 (7.3%)	11 (4.7%)	4 (1.7%)	3 (1.3%)	9 (3.9%)	51 (21.9%)	8 (3.4%)	46 (19.7%)
Middle Age	233	77 (33.0%)	18 (7.7%)	18 (7.7%)	0 (0.0%)	1 (0.4%)	6 (2.6%)	43 (18.5%)	27 (11.6%)	43 (18.5%)
Older Adults	233	47 (20.2%)	8 (3.4%)	10 (4.3%)	1 (0.4%)	1 (0.4%)	3 (1.3%)	28 (12.0%)	8 (3.4%)	127 (54.5%)
Total	932	243 (26.1%)	50 (5.4%)	66 (7.1%)	7 (0.8%)	9 (1.0%)	34 (3.6%)	180 (19.3%)	46 (4.9%)	297 (31.9%)

^aMisc. is short for Miscellaneous

Participants were also asked to answer questions on symptoms of OSA (snoring, stop breathing, gasping for air) and narcolepsy (sleep attacks and weakness following a strong emotion), as well as other sleep disorder symptoms, including morning headaches, EDS, nightmares, and restless leg symptom of unpleasant (creepy crawly) sensations in legs at bedtime (See Table 20).

Table 20

Sleep Disorders Symptoms by Age Group

	OSA Symptoms			Headache ^c	EDS ^d	Narcolepsy		Nightmares	RLS ^e
	Snore (%)	Stop ^a (%)	Gasp ^b (%)			Attack ^e (%)	Weak ^f (%)		
Young Adults	94 (40.3%)	44 (18.9%)	48 (20.6%)	94 (40.3%)	137 (58.8%)	33 (14.2%)	71 (30.5%)	127 (54.5%)	57 (24.5%)
Adults	114 (48.9%)	64 (27.5%)	61 (26.2%)	78 (33.5%)	146 (62.7%)	42 (18.0%)	51 (21.9%)	125 (53.6%)	52 (22.3%)
Middle Age	126 (54.1%)	73 (31.3%)	54 (23.2%)	71 (30.5%)	121 (51.9%)	42 (18.0%)	49 (21.0%)	103 (44.2%)	58 (24.9%)
Older Adults	121 (51.9%)	75 (32.3%)	43 (18.5%)	45 (19.3%)	121 (51.9%)	39 (16.7%)	42 (18.0%)	94 (40.3%)	47 (20.2%)
Total	455 (48.8%)	256 (27.5%)	206 (22.1%)	288 (30.9%)	525 (56.3%)	156 (16.7%)	213 (22.9%)	449 (48.2%)	214 (23.0%)

^aStop refers to stop breathing or holding breath while sleeping.

^bGasp refers to the individual awakening at night gasping for breath.

^cHeadache refers to experiencing a headache upon waking.

^dEDS or Excessive Daytime Sleepiness refers to feeling extremely sleep during the day so much so that the individual finds themselves falling asleep or fighting the urge to fall asleep.

^eAttack refers to a sudden onset of sleep during the day.

^fWeak refers to experiencing muscular weakness following a strong emotion.

^gRLS or Restless Leg Syndrome refers to an unpleasant feeling (creepy crawly) or sensation in the individual's legs at bedtime.

Frequency analyses were performed on additional sleep-related insomnia questions. Specifically, participants were asked if they napped during the day, if they had experienced a previous sleep study, if they considered their insomnia to be problem, and if they had ever received treatment for insomnia (See Table 21).

Table 21

Other Sleep and Insomnia Questions by Age Group

	<i>n</i>	Sleep-Related Questions		Insomnia	
		Previous Sleep Study	Daytime Napping	Insomnia Problem (%)	Insomnia Treatment (%)
Young Adults	233	9 (3.9%)	80 (34.3%)	163 (70.0%)	33 (14.2%)
Adults	233	29 (12.4%)	100 (42.9%)	175 (75.1%)	49 (21.0%)
Middle Age	233	38 (16.3%)	86 (36.9%)	198 (85.0%)	84 (36.1%)
Older Adults	233	62 (26.6%)	116 (49.8%)	210 (90.1%)	98 (42.1%)
Total	932	138 (14.8%)	382 (41.0%)	746 (80.0%)	264 (28.3%)

Participants were also asked to provide information regarding medical and psychiatric comorbidities. For each medical and psychiatric condition, participants were required to answer whether they have had the condition currently or in the past. Regarding the comorbid medical conditions, participants were assessed for epilepsy/serious head injury, cancer, heart disease, kidney disease, liver disease/hepatitis, diabetes, thyroid disease, and nocturia (See Table 22).

Table 22

Medical Conditions Present by Age Group

	TBI/Epilepsy ^a		Cancer ^b		CVD ^c		Kidney ^d		Hepatitis ^e		Diabetes ^f		Thyroid ^g		Noc/Pros ^h	
	P ⁱ	C ⁱ	P	C	P	C	P	C	P	C	P	C	P	C	P	C
YA ^j	15 (6.4%)	0 (0.0%)	2 (0.9%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	2 (0.9%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	5 (2.1%)	0 (0.0%)	6 (2.6%)	0 (0.0%)	7 (3.0%)
A	15 (6.4%)	3 (1.3%)	1 (0.4%)	1 (0.4%)	0 (0.0%)	5 (2.1%)	1 (0.4%)	2 (0.9%)	2 (0.9%)	0 (0.0%)	3 (1.3%)	2 (0.9%)	5 (2.1%)	16 (6.9%)	1 (0.4%)	21 (9.0%)
MA	25 (10.7%)	4 (1.7%)	22 (9.4%)	2 (0.9%)	4 (1.7%)	11 (4.7%)	2 (0.9%)	3 (1.3%)	6 (2.6%)	9 (3.9%)	4 (1.7%)	22 (9.4%)	5 (2.1%)	27 (11.6%)	2 (0.9%)	33 (14.2%)
OA	17 (7.3%)	2 (0.9%)	44 (18.9%)	11 (4.7%)	18 (7.7%)	21 (9.0%)	0 (0.0%)	9 (3.9%)	10 (4.3%)	7 (3.0%)	5 (2.1%)	36 (15.5%)	6 (2.6%)	31 (13.3%)	11 (4.7%)	72 (30.9%)
Total	72 (7.7%)	9 (1.0%)	69 (7.4%)	14 (1.5%)	22 (2.4%)	37 (4.0%)	5 (0.5%)	15 (1.6%)	18 (1.9%)	16 (1.7%)	12 (1.3%)	65 (7.0%)	16 (1.7%)	80 (8.6%)	14 (1.5%)	133 (14.3%)

^aTBI/Epilepsy assesses if the participants have experienced a TBI or Seizure in the past or present.

^bCancer assesses if the participants have cancer in the past or at present. There is no assessment of type of cancer.

^cCVD references Cardiovascular Disease and if the individual has experienced it in the past or at present.

^dKidney refers to Kidney Disease.

^eHepatitis assesses for any type of Hepatitis. As with cancer, there is no assessment of type.

^fDiabetes encompasses Type I and Type II Diabetes.

^gThyroid assesses for any thyroid-related disease and does not assess for specific diagnoses.

^hNoc/Pros refers to Nocturia or Prostate concerns where an individual may be required to awaken several times during the night due to the need to urinate.

ⁱP and C refer to Past or Current, indicating the time at which the individual experienced/is experiencing these medical concerns.

^jThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Regarding comorbid psychiatric conditions, participants were assessed for MDD, anxiety disorders, obsessive compulsive disorder, schizophrenia, bipolar disorder or manic depression, and severe memory problems (identified as dementia). Additionally, participants were asked whether they had ever experienced inpatient psychiatric hospitalizations (See Table 23). Participants were also asked about use of psychiatric medications and family psychiatric history (See Table 24).

Table 23

Psychiatric Conditions Present by Age Group

	Depression		Anxiety		OCD ^a		Schizophrenia		Bipolar ^b		Memory ^c		Psych. Hosp. ^d	
	P ^e	C ^e	P	C	P	C	P	C	P	C	P	C	P	C
YA ^f	34 (14.6%)	68 (29.2%)	19 (8.2%)	64 (27.5%)	7 (3.0%)	5 (2.1%)	1 (0.4%)	0 (0.0%)	7 (3.0%)	12 (5.2%)	4 (1.7%)	6 (2.6%)	28 (12.0%)	1 (0.4%)
A	40 (17.2%)	60 (25.8%)	21 (9.0%)	50 (21.5%)	0 (0.0%)	8 (3.4%)	0 (0.0%)	1 (0.4%)	6 (2.6%)	13 (5.6%)	2 (0.9%)	3 (1.3%)	30 (12.9%)	0 (0.0%)
MA	45 (19.3%)	53 (22.7%)	20 (8.6%)	45 (19.3%)	2 (0.9%)	3 (1.3%)	0 (0.0%)	3 (1.3%)	2 (0.9%)	11 (4.7%)	3 (1.3%)	11 (4.7%)	17 (7.3%)	1 (0.4%)
OA	40 (17.2%)	49 (21.0%)	17 (7.3%)	41 (17.6%)	1 (0.4%)	4 (1.7%)	2 (0.9%)	2 (0.9%)	1 (0.4%)	5 (2.1%)	4 (1.7%)	7 (3.0%)	16 (6.9%)	1 (0.4%)
Total	159 (17.1%)	230 (24.7%)	77 (8.3%)	200 (21.5%)	10 (1.1%)	20 (2.1%)	3 (0.3%)	6 (0.6%)	16 (1.7%)	41 (4.4%)	13 (1.4%)	27 (2.9%)	91 (9.8%)	3 (0.3%)

^aOCD refers to Obsessive Compulsive Disorder

^bBipolar refers to either Bipolar I Disorder or Bipolar II Disorder. There is no assessment for specific type.

^cMemory refers to any severe memory issues the patient may have experienced in the past or at present.

^dPsych. Hosp. refers to Psychiatric Hospitalization and the question asks if the individual has ever received treatment or is currently receiving treatment from a psychiatric hospital.

^eP and C refer to Past or Current, indicating the time at which the individual experienced/is experiencing these psychiatric concerns.

^fThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Table 24

Other Psychiatric-Related Questions by Age Group

	Other Psychiatric Questions	
	Psych. Med. ^a (%)	Fam. Psych. Hx ^b (%)
Young Adults	95 (40.8%)	70 (30.0%)
Adults	97 (41.6%)	54 (23.2%)
Middle Age	68 (29.2%)	32 (13.7%)
Older Adults	65 (27.9%)	37 (15.9%)
Total	325 (34.9%)	193 (20.7%)

^aThese numbers reflect the number of individuals in each age group who endorsed ever taking psychiatric medication.

^bThese numbers represent the number of individuals who endorsed a family history of psychiatric difficulties.

CHAPTER 5: DISCUSSION

The present study primarily examined age group differences for SCDs, sleep-related daytime dysfunction, and problem endorsement; that is, the self-reported perception that SCD and sleep-related daytime dysfunction are problems. The age groups assessed in this study were YAs (18 to 29 years), As (30 to 44 years), MAs (45 to 64 years), and OAs (65 to 89 years). Previous research found age differences for sleep disturbances and sleep-related daytime dysfunction (Dijk et al., 2010; Morin et al., 1993; Ohayon et al., 2004); however, a literature review revealed no research assessing problem endorsement of SCD or any study evaluating age group differences for these endorsed problems. This gap in the literature may be due to researchers assuming that the presence of SCD constitutes a problem for the individual. The literature also suggests that patients and clinicians alike may dismiss these SCD as a “normal” and acceptable part of aging and not assess further (Mander et al., 2017).

Furthermore, the present study evaluated both SCD and sleep-related daytime dysfunction, whereas much of the existing literature focuses on either one of these two categories. There have been several studies that have assessed both SCD and sleep-related daytime dysfunction; however, many fall under the category of meta-analyses rather than true experimental designs (Ancoli-Israel & Martin, 2006; Ohayon et al., 2004). Additionally, many sleep studies examine sleep across a very limited age range, thus restricting the scope of findings and generalizability of the results. Finally, there are no previous studies that inquired explicitly about problem endorsement for each SCD of sleep latency (SL), nighttime awakenings (NWAK), wake after sleep onset (WASO), early morning awakenings (EMA), or total sleep time (TST). These shortcomings in the

existing literature translate to significant limitations within the field of insomnia research and clinical practice, calling into question recommendations for evidence-based assessment and treatment of sleep concerns within the field of sleep medicine, particularly regarding populations varying in age. Furthermore, as currently practiced, sleep assessment and treatment recommendation are often not a consistently or comprehensively informed by patients' medical health (crucial psychiatric, neurological, cardiovascular, and hormonal functions), despite how relevant this is to sleep (Grandner & Malhotra, 2015).

As such, the goal of the present was to identify age group differences in SCD, sleep-related daytime dysfunction, and problem endorsement in a sample of individuals between the ages of 18 and 89 years. This study attempted to address the aforementioned limitations of the extant sleep literature and the results of this study are intended to inform assessment and treatment of adult insomnia within the aforementioned age groups, with particularly interesting findings on OAs (65 to 89 years).

Findings and Clinical Implications

As expected, SCD worsens with age (except for SL). Sleep-related daytime dysfunction did not worsen with age, and for the one variable that showed an age-related effect (concentration), there was a negative relationship in that older age groups endorsed less concentration difficulties. Finally, problem endorsement, contrary to expectation, increased with age (except for SL).

SCD and problem endorsement. Ultimately, results indicated that there were age group differences for NWAK, about which MAs reported at the highest frequency ($M = 3.1$). The results also indicated age group differences for WASO and EMA, for which

OAs reported the longest length of WASO ($M = 44.71$) and EMA ($M = 72.33$) in minutes. There were group differences for TST, for which YAs reported the longest ($M = 373.39$) and MAs reported the shortest TST ($M = 334.76$) in minutes. There were no age group differences for SL.

As such, the results indicate age group differences for WASO insomnia, EMA insomnia, and overall TST, especially within adults over the age of 45 years. Furthermore, there were clear trends in NWAK and TST, implying slight improvement in adults over the age of 65 years. This finding directly contradicts existing literature that suggested OAs experience the greatest SCD overall (Ohayon et al., 2004).

Furthermore, the results indicated significant relationships for age groups and problem endorsement of NWAK, WASO, and TST. Specifically, problem endorsement for these SCDs peaked for MAs (NWAK [81.3%], WASO [93.5%], and TST [78.1%]) before declining slightly in the OA group (NWAK [79.0%], WASO [88.1%], and TST [76.4%]). There were also significant linear relationships for age groups and problem endorsement of EMA, for which OAs showed highest percent problem endorsement (82.7%). There were no significant differences for SL.

As such, the results indicated that MAs and OAs had the highest problem endorsement for all SCD, except SL. This is inconsistent with the original hypothesis, which posited that older age groups (MAs and OAs) would be less likely to report their SCD symptoms as problematic. Of note, the original hypothesis was based on literature that stated OAs are less likely to perceive these SCD as problematic (Ohayon et al., 2004). As the results contradict a well-established meta-analysis, it is essential that

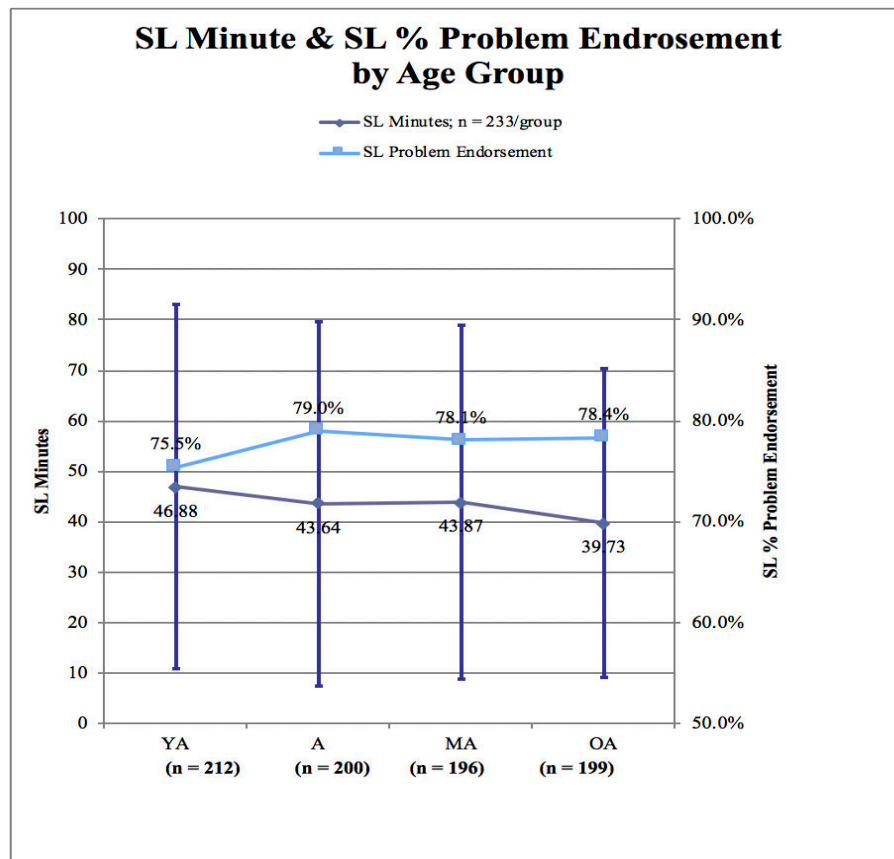
closer attention be paid, clinically, to MAs and OAs, as they are the groups (within this sample) to most likely endorse SCDs as problems.

Combined results. Combined graphs were used to assess how SCD duration/frequency mapped onto SCD problem endorsement. Specifically, these graphs were created to visually compare how SCD and problem endorsement of SCD changed across age groups.

Inconsistent with the existing literature (e.g., Ohayon et al., 2004), the current study found that self-reported SL did not vary significantly by age cohort, either by duration or problem endorsement. There was no linear or curvilinear trend for minutes of SL or problem endorsement. This indicated that age groups reported similar duration of SL. The findings also suggested that age groups have similar perceptions in regard to the problem endorsement of SL. This is inconsistent with the existing literature that found significant increases in SL as individuals age (Ohayon et al., 2004). The findings of the present study suggest that clinicians should continue to assess for sleep initiation difficulties and endorsement of these problems across the lifespan (See Graph 17).

Graph 17

Sleep Latency (in Minutes) and Sleep Latency Percent Problem Endorsement by Age Group



Note: SL Minutes by Age Group: OA = MA = A = YA, $p = .159^a$

SL Problem Endorsement by Age Group: OA = MA = A = YA, $p = .832$

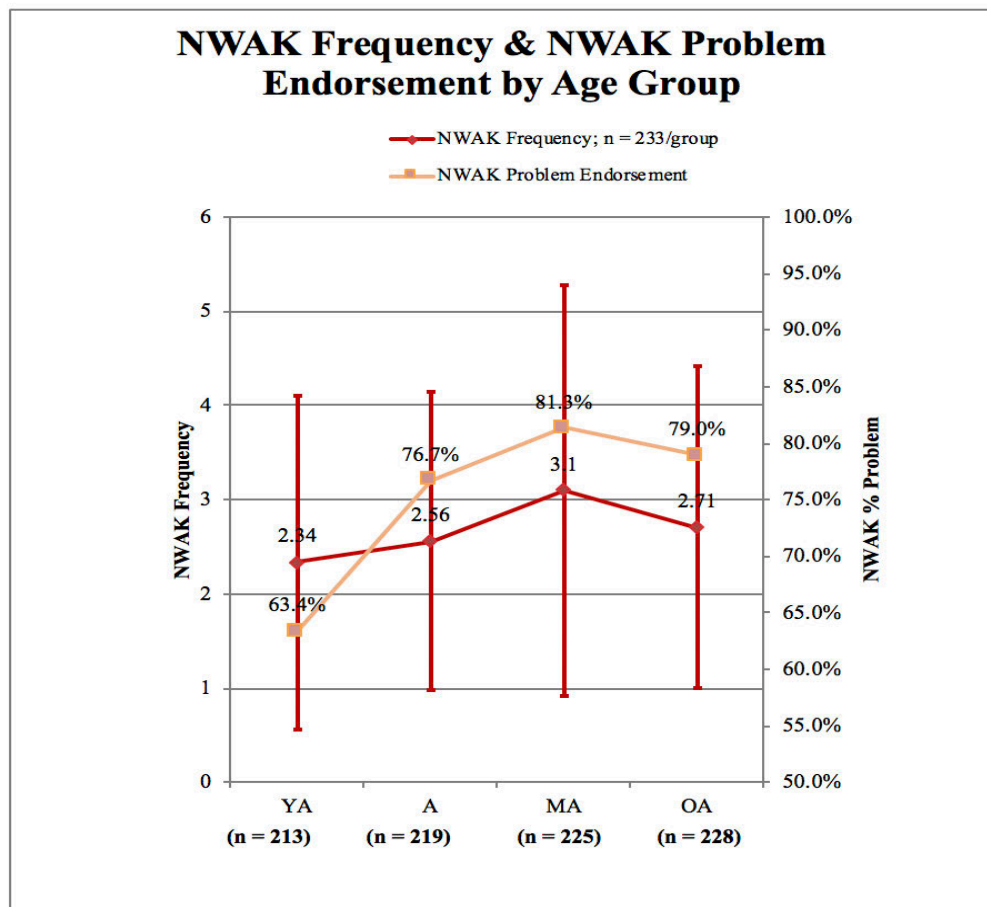
^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Regarding NWAK, the present study found significant age group differences in which MAs reported the highest frequency of NWAK (81.3%). This suggests that NWAK does not become progressively worse—in frequency or problem endorsement—across age groups, but peaks at MA (see Graph 18). YAs as a group differed from MAs in that they experienced a fewer number of NWAK and, thus, fewer individuals endorsed

NWAK as a problem. There were minimal differences between the MA and OA groups in regard to frequency of NWAK and NWAK problem endorsement. This is consistent with the existing literature in that OAs have less in-group variability regarding NWAK (Dillon et al., 2014).

Graph 18

Nighttime Awakenings (Frequency) and Nighttime Awakenings Percent Problem Endorsement by Age Group



Note: NWAK Frequency by Age Group: $OA = (MA > A = YA)$, $p < .001^a$

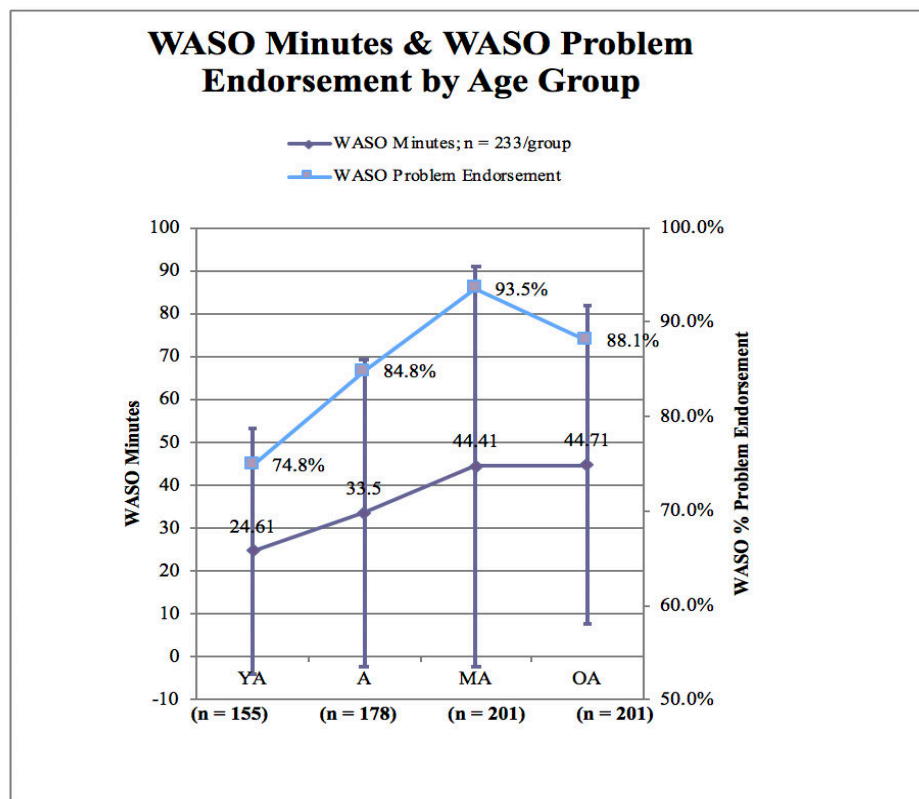
NWAK Problem Endorsement by Age Group: $MA > OA > A > YA$, $p < .001$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

In terms of WASO, MAs had significantly longer WASO ($M = 44.41$) and higher problem endorsement of WASO (93.5%) than YAs ($M = 24.61$; 74.8%). There appears to be a linear trend regarding WASO length and problem endorsement (see Graph 19). It is notable that this trend peaks for MAs and decreases again for OAs, which is inconsistent with the findings in existing literature, stating that WASO increases with age (Dillon et al., 2014; Ohayon et al., 2004).

Graph 19

Wake After Sleep Onset (in Minutes) and Wake After Sleep Onset Percent Problem Endorsement by Age Group



Note: WASO Minutes by Age Group: (OA = MA) > A > YA, $p < .001^a$

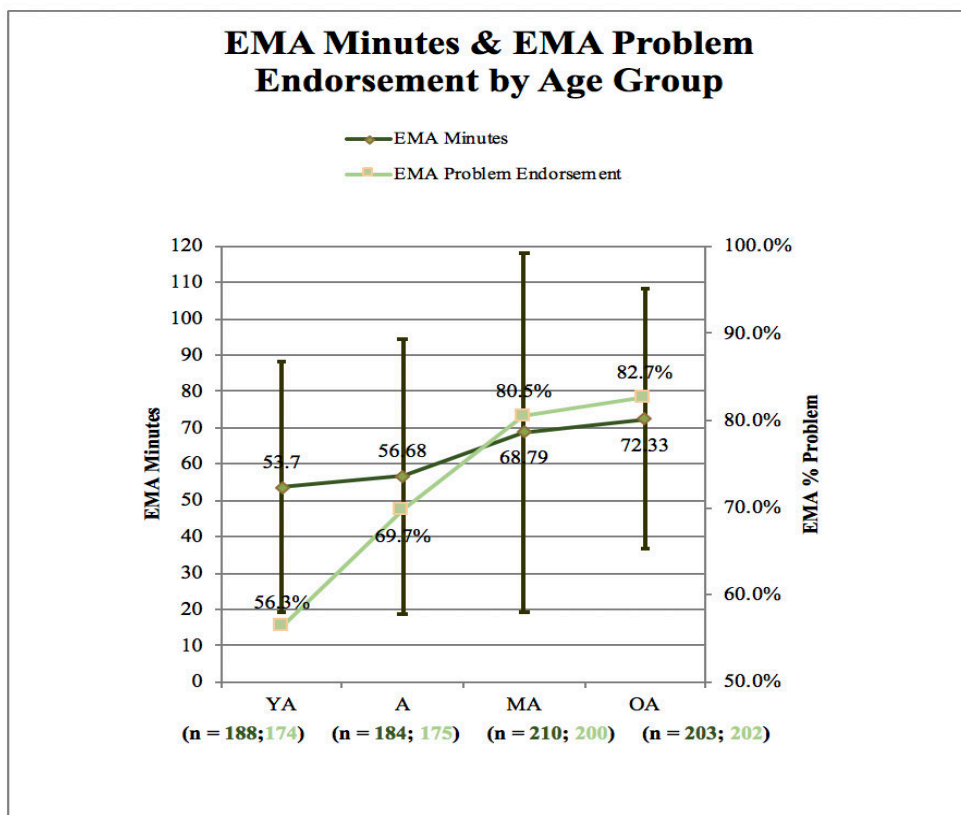
WASO Problem Endorsement by Age Group: MA > OA > YA > A, $p < .001$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

Furthermore, EMA (minutes and problem endorsement frequency) varied by age in that OAs ($M = 72.33$) reported being awake significantly earlier than YAs ($M = 53.70$) (see Graph 20). Additionally, problem endorsement of EMA also peaked in the OA group (82.7%) and is significantly greater than problem endorsement of EMA in the YA group (56.3%), a finding that is consistent with the existing literature (Ohayon et al., 2004).

Graph 20

Early Morning Awakenings (in Minutes) and Early Morning Awakenings Percent Problem Endorsement by Age Group



Note: EMA Minutes by Age Group: (OA = MA) > (A = YA), $p < .001^a$

EMA Problem Endorsement by Age Group: OA > MA > A > YA, $p < .001$

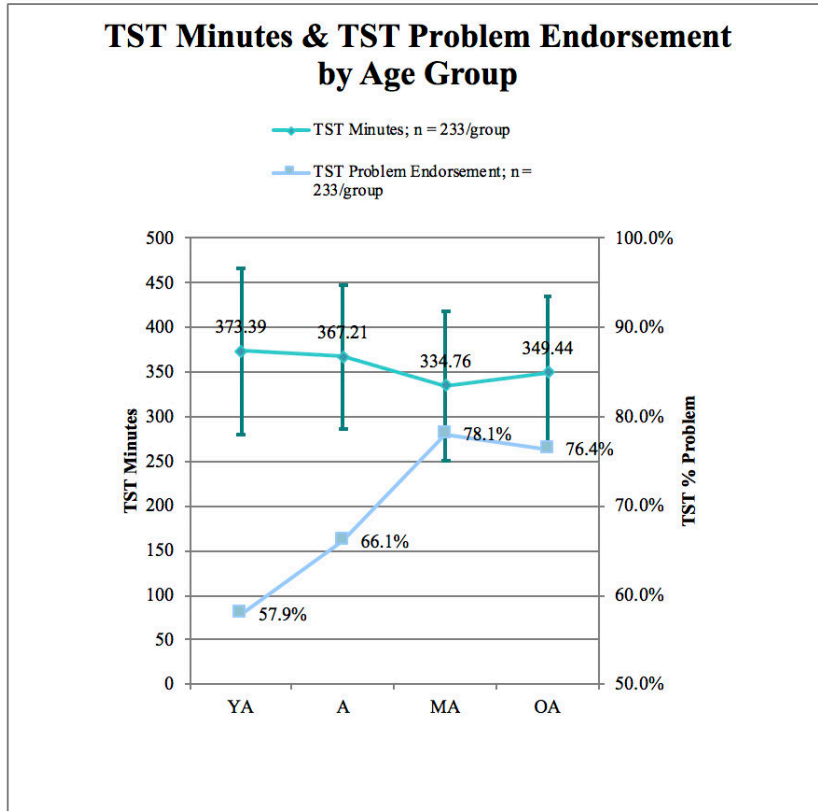
^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

The present study also found that problem endorsement of NWAK, WASO, and EMA varied significantly by age cohort and that this was especially true for WASO and EMA. The across age cohorts effects seems greatest for EMA. Specifically, the present study found significant differences in some age groups regarding the endorsement of WASO and EMA as problems. These differences for WASO and EMA were especially apparent in YAs (WASO = 74.8%; EMA = 56.3%), As (WASO = 84.8%; EMA = 69.7%), and OAs (WASO = 88.1%; EMA = 82.7%). There were no significant differences in age groups who endorsed WASO and EMA as problems, in percent of MAs (WASO 93.5%; EMA = 80.5%) and OAs (WASO = 88.1%; EMA = 82.7%). This suggests that adults above the age of 45 similarly report problem endorsement for WASO and EMA and should receive careful assessment from their health care providers regarding these SCD, regardless of age.

Regarding TST (minutes and problem endorsement), the present study found that these variables differed with age. The percent complaint presented a mirror image of TST length (see Graph 21). The lowest TST problem endorsement was by the YA group (57.9%) and significantly rose to peak for MAs (78.1%). Following this peak, there was a slight non-significant decrease for OAs (76.4%). These findings are notable in that TST is often not an accurately calculated variable (e.g., $TIB - [SL + WASO + EMA] = TST$), but rather an estimated number by the participant. This is especially interesting because TST length is at a range of 334.76 minutes at the lowest (MA) and 373.39 minutes at the highest (YA). The difference between these two values is 38.63 minutes and an effect size of 0.437, indicating a small effect size.

Graph 21

Total Sleep Time (in Minutes) and Total Sleep Time Percent Problem Endorsement by Age Group



Note: TST Minutes by Age Group: OA = (MA < A = YA); OA < YA, $p < .001^a$

TST Problem Endorsement by Age Group: MA > OA > A > YA, $p < .001$

^aThe abbreviations represent the different age groups as follows: Young Adult (YA); Adult (A); Middle Aged (MA); and Older Adult (OA).

In contrast to self-reported TST, percent of problem endorsement for TST appears to drastically worsen between YAs and MAs, with a 35.4% increase in problem endorsement. As such, there is not a large amount of variability with respect to severity but there is with respect to problem endorsement. This allows for the development of several theories. For example, it may potentially be a “threshold” concern, for which under a certain length of TST, the ability to adapt is initially poor but worsens with age (at least until age 65). Again, these findings are consistent with the existing literature, which has stated that TST may decrease with age; however, it is inconsistent with the literature that has shown that older age groups are less likely to endorse SCD as problematic (Mander et al., 2017; Ohayon et al., 2004).

There are many possible explanations for these results. One particularly plausible possibility is that SCD exacerbates, or is perceived to exacerbate, other comorbidities, and that these comorbidities tend to increase with age. In other words, SCD in general exacerbates with comorbid disorders and as people age, they are susceptible to an increasing number of comorbid disorders. This is especially true for OAs. As such, the presence of comorbidities may be causing people to identify SCD severities as more problematic.

Alternatively, there may be a reason that OAs consistently reported problem endorsement of SCD at a lower rate than MAs, except for EMA. For example, OAs may be healthy survivors (in terms of disease survivorship) or are used to being unhealthy. This theory aligns with the existing literature, which supports the idea that chronic SCDs can lead to, for instance, mild cognitive impairment immediately, and potentially comorbid medical, neurological, endocrine, and/or psychiatric disorders over time if left

untreated. In particular, prolonged poor sleep can lower the threshold for medical and psychiatric illnesses, such as cardiovascular disease, diabetes, dementia, obesity, stroke, anxiety, and depression (Grandner, 2012). As such, it is paramount that all age groups be assessed for SCD and problem endorsement, even if the patient or clinician attributes these symptoms to “normal aging” (Mander et al., 2017).

Sleep-related daytime dysfunction. The study also assessed age group differences for symptoms of sleep-related daytime dysfunction. Out of the five domains assessed (self-reported concentration, restedness, impaired daytime function, fatigue, and daytime sleepiness), there were only concentration age effects, wherein OAs (74.3%) reported significantly less concentration difficulties than YAs (57.1%). The results suggest that endorsement of concentration difficulties varies by age, with a percent decrease over time of -23.3% between the age groups of YA and OA.

Otherwise, self-reports of feeling rested upon waking, impaired daytime function, and daytime sleepiness did not vary by age. Additionally, the total reports of sleep-related daytime dysfunction did not significantly differ by age group. These significant concentration findings and the lack of findings within the other domains are inconsistent with the existing literature that has shown a relationship between poor sleep and diminished cognitive functioning in adults over the age of 65 (Foley et al., 1995; Schmutte et al., 2007). It is also inconsistent with existing literature that has shown a curvilinear relationship between tiredness and age (Grandner, Martin, et al., 2012).

It is important to note that the existing literature has attributed the diminished daytime and cognitive functioning of older adults to both a decline in sleep and a decline in overall health (Foley et al., 1995; Grandner, Martin, et al., 2012). It is possible that the

present study would have had more robust findings if it had taken medical and psychiatric comorbidities into account when assessing for sleep-related daytime functioning.

Nevertheless, it may still be the case that older individuals perceive these sleep-related daytime dysfunctions as “normal” and acceptable in the face of medical and psychiatric comorbidities.

Implications for clinical settings. As always, what researchers discover varies based on what they are looking at. It is true that SCD worsens with age and that people, as they age, increasingly view this as problematic. It is also true that they view these SCDs as problems regardless of the effects on daytime function. Put differently, whether something is a problem varies but how the question is asked: Is it a problem because of its consequences? Is it a problem merely because of its occurrence? The answers may differ. As such, this study illuminated an additional question that should be applied to sleep assessments: Is this a problem?

Based on the results of this study and the breadth of literature supporting the negative impact of chronic poor sleep, it is essential that primary care physicians and health care professionals within outpatient behavioral health care settings perform effective evaluations of each individual's sleep, even if that individual does not endorse their overall sleep as being problematic. These settings are perfectly placed for more individuals to receive evaluation regarding their sleep. An in-depth sleep assessment on the part of the clinician would provide more insight into the patient's overall physical health, mental health and health quality of life. Furthermore, it may inform the clinician's decision to refer the patient for cognitive behavioral treatment and provide additional clarification for the reasons for referral.

Assessment. Specific questions about length and frequency of SL, NWAK, WASO, EMA, and TST may provide some much needed insight into the pattern of a patient's sleep. Additionally, although an individual may not be dissatisfied with their overall sleep, he or she may find one or two aspects of SL, NWAK, WASO, EMA, and TST to be of concern. Thus, primary care physicians and other clinicians should inquire about these specific aspects of sleep. This may be especially true for older individuals who, as research suggests, are less likely to endorse their overall sleep as a problem (Grandner, Martin, et al., 2012; Grandner, Patel, & Gooneratne, 2012). This was further evidenced by the results of the present study, which showed that there were no significant differences between MAs and OAs for NWAK, WASO, EMA, and TST; however, OAs were less likely than MAs to endorse these SCDs as problems.

It should be noted that this online survey is not a substitute for a health care provider. As such, clinicians should be aware that even when they inquire as to whether patients view their SCDs or sleep-related daytime dysfunction to be problems, a lack of problem perception may obscure clinically significant concerns. There are a number of valid and reliable measures that can serve as a basic format should a clinician wish to expand their evaluation of their patient's sleep beyond the general inquiry of, "How has your sleep been?" For example, should the patient express difficulty falling asleep, it may be beneficial to administer the ISI (Morin, 1993). Additionally, if there is suspicion of other sleep disorders, it may also be wise to administer the SDS-CL-25 to obtain a comprehensive review of sleep disorder symptoms for 25 different disorders (Klingman et al., 2017). Clinicians may also use these subjective sleep measures to determine what to follow up on in regard to problem endorsement.

It is important also for clinicians to remember that these problem endorsements may vary by age and that sleep concerns are not necessarily an inherent part of the aging process. The results support that, even if participants do not *report* sleep concerns, they still may *experience* significant deviations from healthful sleep, specifically NWAK, WASO, EMA, TST, and their ability to concentrate during the day. Notably, the present study found age-related differences. Consequently, it would be important for clinicians to become familiar with these age-related differences and consider them when evaluating their patients and making treatment recommendations.

Many individuals who report SCDs and are dissatisfied with their sleep also report some level of comorbidity or poor overall health (Stephen et al., 2017). As individuals age, they are more likely to experience additional medical, neurological, and psychological concerns (Grandner, 2012); however, research also suggests that subjective age plays a role in how healthy individuals feel overall (Stephen et al., 2017). As such, it may also be beneficial for a clinician to inquire about a patient's subjective and chronological age, as research has indicated that subjective age can be a biopsychosocial marker of aging and, therefore, an accurate marker of an individual's risk for worsened sleep quality (Stephen et al., 2017).

In sum, regardless of reported SCD, problem endorsement, sleep-related daytime dysfunctions, chronological age, subjective age, or comorbidities, comprehensive assessment of sleep should be performed within health care settings. In-depth sleep-related questions are particularly important because they may inform treatment planning and the decision to refer an individual for evidence-based insomnia treatment, specifically CBT-I.

Treatment. CBT-I is considered the first line of treatment for insomnia disorder by the American College of Physicians. When compared to individuals receiving a placebo treatment or pharmacotherapy, those who engage in CBT-I have shown a significantly greater success rate, an element that remains apparent even at follow-up (Okajima, Komada, & Inoue, 2011). In order to refer patients for this treatment, health care providers must become more educated in sleep medicine and develop adequate assessments that reinforce their referral decisions. An extensive sleep assessment would also benefit the behavioral sleep medicine specialist, as the reason for referral would become clearer.

Limitations

Despite the findings and implications for the treatment of insomnia in adults, particularly elders, this study is not without its limitations. A major limitation of this study is the exclusive use of retrospective self-report measures, which may not be as accurate as more objective measures. Retrospective self-report of a single time point is usually flawed due to subjectivity and reliance on retrospective memory of sleep experiences, which may be inaccurate (Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002). This may especially be the case with TST, in that TST is generally one of the least accurately calculated variables because calculated TST (cTST) can be found through the following equation if the clinician has the correct variables: $TIB - (SL + WASO + EMA) = cTST$. Instead, self-reported TST often requires an estimate, usually without consideration or even awareness of the aforementioned TST components. Furthermore, because the questionnaires were presented online, there were no resources

available for clarification of questions. This could constitute an artifact and threat to validity and reliability, in that participants may have resorted to guessing.

An additional limitation is that the data are from a community-based sample, primarily made up of individuals who self-selected to complete this online survey because they reported experiencing sleep concerns. Due to the potential for volunteer-response bias, there are very few potential “good sleepers” with whom to compare the individuals reporting SCD and, thus, there is no control group. It is, however, important to note that much of the sleep literature consists of studies comparing poor sleepers and good sleepers (Lichstein, Durrence, D. J. Taylor, Bush, & Riedel, 2003). Few studies have focused solely on poor sleepers and their problem endorsements in regard to their sleep.

Another limitation within this study was that in the MANOVA, the Levene’s test was found to be significant for the variables of WASO, EMA, and TST. As such, equal variances for these variables cannot be assumed across groups and the results of the MANOVA for these variables in particular are unlikely to have occurred from random sampling. This could be due to the number reasons. For example, although collected in the original survey, many mediating and moderating variables were not taken into account. As individuals age, they are likely to experience new medical, neurological, and psychological diagnoses. Although assessed in the www.sleeplessinphilly.com survey, these variables were not considered during the statistical analyses. As such, the findings in this study did not account for these potentially confounding variables.

Finally, there are some gaps within the demographic questions in the www.sleeplessinphilly.com measure. First, the sex question only allowed for binary

options. As such, individuals may have been forced to choose a sex with which they do not fully identify in order to complete the survey. If the survey were to be adapted, a third option of “other” would be required, at the very minimum. Second, the race question provided very limited options and did not allow for additional race choices, such as Middle Eastern or Mixed Race. Third, the questionnaire did not evaluate subjective age (how old an individual “feels”), marital status, type of residence, number of individuals in the household, number of children (if any), or whether the individual consistently had a bed partner, all of which have been empirically shown to impact sleep. Assessing these factors may have provided more data to differentiate individuals who endorsed SCD from those who did not.

Future Research

Resiliency, survivorship, and cumulative morbidity. Grandner, Patel, and Gooneratne (2012) proposed the concept of survivorship, in that individuals with severe sleep disturbances in younger age groups may not survive to older age groups. Similarly, Grandner, Patel, et al., also postulated that individuals with very poor health are not likely to survive to older age and, that those who do survive have developed a degree of resilience to further health decline. Research has yet to adequately address this concept.

As this information is already collected in the present dataset, it may be beneficial for the researchers to perform additional statistical analyses that take into consideration the plethora of medical, neurological, and psychological comorbidities present in the entire sample. Specifically, it may also be worth analyzing whether there is an increase of these comorbidities over the lifespan and how these relate to SCD, sleep-related daytime dysfunction, problem endorsement, or sleep disorder symptoms.

In regard to sleep disorder symptoms, it would be interesting to evaluate age group differences for each symptom. A preliminary analysis determined significant age-related differences in OSA symptoms (snoring, breathing pauses, gasping for air), morning headaches, muscle weakness, and nightmares. This abstract is available upon request (Boyle et al., 2019). It would be interesting to update these analyses with more participants or to explore the established findings even further by controlling for medical comorbidities.

Additionally, variables that were assessed may also benefit from a more in-depth evaluation. Specifically, the variable of BMI was profound in that most individuals fell into the category of overweight (25 to 29.9 kg/m²; 40.8%), followed by the obese category (≥ 30 kg/m²; 31.3%). The literature has suggested that increased BMI can lead to anatomic alterations that leave individuals susceptible to developing obstructive sleep apnea (Somers et al., 2008). Furthermore, there is extensive research indicating a clear relationship between sleep duration and quality, obesity, and metabolic health (Grandner et al., 2015). As such, it may be worth performing additional covariate analyses to determine the impact of BMI on the present results.

Ethnic/racial disparities. In general, short sleep (5 to 6 hours) and long sleep (> 9 hours) duration is associated with an increased risk of cardiovascular disease and other health concerns. Whinnery et al. (2014) developed a study to determine whether the risk of short or long sleep duration is mediated by differences in race or ethnicity. The researchers collected self-report sleep duration data in Black/African Americans, Hispanic/Latino Americans, Asian Americans, and White Americans. The data indicated that Black/African Americans were three times more likely to report short (5 to 6 hours)

or very short (< 5 hours) sleep as compared to White Americans (Whinnery, Jackson, Rattanaumpawan, & Grandner, 2014). Overall, the researchers found that non-White individuals were more likely to report short or very short sleep as compared to White individuals, thus placing these populations at a higher risk for cardiometabolic disease (Whinnery et al., 2014). As such, more research should be considered for ethnic and racial disparities, daytime dysfunction, and future treatment strategies to address specific stressors. Specifically, it would be interesting to expand upon these results by assessing TST in Black/African American participants as compared to White participants within the dataset used in the present study.

Gender disparities. The existing literature has also shown gender disparities in sleep disturbances and sleep quality. For example, one article indicated that African American women are the most likely group to experience insomnia at some point in their lives (Grandner et al., 2010). Another study found that women reported more sleep disturbances, more symptoms of sleep-related daytime dysfunction, and more somnolence than men (Grandner, Martin, et al., 2012). Additional changes may be exhibited following aging and the onset of menopause (Garland et al., 2018). It would be particularly interesting to utilize the female-specific questions within the www.sleeplessinphilly.com (Items 90 through 94) to compare SCD and SRDD while also controlling for variables such as birth control use, pregnancy, and menopause (see Appendix B).

Napping. It has been shown that individuals with insomnia or SCD try to compensate for their lack of sleep. One example previously mentioned as a compensatory behavior is napping. It is also likely that individuals with insomnia may

not be able to nap, but there may be additional factors that make napping more likely. Factors that may make napping more likely include the presence of another comorbid sleep disorder, such as OSA or narcolepsy; significant EDS that leads to unplanned napping; or age of the individual. Age in particular is associated with unplanned napping (Foley et al., 2007).

In regard to the results of the present study, the age group with the highest number of endorsed naps was OAs (49.8%), followed by As (42.9%), MAs (36.9%), and YAs (34.3%). It may be worth running future analyses to determine whether there were significant differences in napping behavior by age group; however, it cannot be determined whether these reported naps were planned or unplanned, as the questionnaire did not require the participants to specify.

Furthermore, it may be interesting to perform additional analyses comparing the sample of individuals who endorsed napping to the rest of the sample. Specifically, it may be helpful to determine whether there were significant age-related differences and whether the factors mentioned above that increase the likelihood of napping can predict napping behavior. It may also be interesting to determine whether there is a direct correlation between napping behavior and EMA.

Conclusion

The United States continues to have the highest economic burden in the world related to insufficient sleep (between \$280 and \$411 billion per year) and researchers predict that this will increase to at most \$456 billion by 2030 (Hafner et al., 2017). What is most concerning is that SCDs, sleep-related daytime dysfunctions, and problem endorsements could have been assessed in their earlier stages prior to the onset of full

disorders. This is concerning because the literature suggests that poor sleep is related directly to the exacerbation of medical, neurological, and psychological disorders (Grandner & Malhotra, 2015). As such, the onus to provide extensive sleep assessment falls on the shoulders of clinicians. This is because individuals, especially as they age, may be less inclined to report their SCDs and sleep-related daytime dysfunctions as a problem to their health care providers (Grandner, Patel, et al., 2012; Ohayon et al., 2004). This may be due to the misconception that a decline in sleep quantity and sleep quality is a “normal” and acceptable sign of aging (Mander et al., 2017).

Furthermore, current methodology of sleep assessment is generally not a consistent or comprehensive measure of health or sleep pattern, despite how closely intertwined sleep is with medical, neurological, and psychological concerns (Grandner & Malhotra, 2015). The results of this study provide insight into the variations in age groups of self-reported poor sleepers, which health care providers can use to more carefully assess and plan treatment. Additionally, this study provides guidelines about the questions these providers should use when assessing sleep in their patients. Specifically, they should assess SCDs, sleep-related daytime dysfunction and—as importantly—whether the individual considers these SCD and sleep-related daytime dysfunction to be problematic. It is hoped that the results of this study will inform health care providers to allow them to more accurately assess and treat insomnia and SCD.

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APPENDIX A

Categorization of Current Literature

Daytime Dysfunction	Sleep Continuity Disturbances
Garland, S. N., Rowe, H., Repa, L. M., Fowler, K., Zhou, E. S., & Grandner, M. A. (2018). A decade's difference: 10-year change in Insomnia symptom prevalence in Canada depends on sociodemographics and health status. <i>Sleep health</i> , 4(2), 160-165.	Almeida, O. P., & Pfaff, J. J. (2005). Sleep complaints among older general practice patients: association with depression. <i>Br J Gen Pract</i> , 55(520), 864-866.
Morin, C. M., Stone, J., Trinkle, D., Mercer, J., & Remsberg, S. (1993). Dysfunctional beliefs and attitudes about sleep among older adults with and without Insomnia complaints. <i>Psychology and aging</i> , 8(3), 463.	Schmutte, T., Harris, S., Levin, R., Zweig, R., Katz, M., & Lipton, R. (2007). The relation between cognitive functioning and self-reported sleep complaints in nondemented older adults: results from the Bronx aging study. <i>Behavioral sleep medicine</i> , 5(1), 39-56.
Foley, D. J., Vitiello, M. V., Bliwise, D. L., Ancoli-Israel, S., Monjan, A. A., & Walsh, J. K. (2007). Frequent napping is associated with excessive daytime sleepiness, depression, pain, and nocturia in older adults: findings from the National Sleep Foundation '2003 Sleep in America' Poll. <i>The American journal of geriatric psychiatry</i> , 15(4), 344-350.	Foley, D. J., Monjan, A. A., Brown, S. L., Simonsick, E. M., Wallace, R. B., & Blazer, D. G. (1995). Sleep complaints among elderly persons: an epidemiologic study of three communities. <i>Sleep</i> , 18(6), 425-432.
Waller, K. L., Mortensen, E. L., Avlund, K., Osler, M., Fagerlund, B., Lauritzen, M., & Jennum, P. (2016). Subjective sleep quality and daytime sleepiness in late midlife and their association with age-related changes in cognition. <i>Sleep medicine</i> , 17, 165-173.	Dijk, D. J., Groeger, J. A., Stanley, N., & Deacon, S. (2010). Age-related reduction in daytime sleep propensity and nocturnal slow wave sleep. <i>Sleep</i> , 33(2), 211-223.
Vahtera, J., Westerlund, H., Hall, M., Sjösten, N., Kivimäki, M., Salo, P., ... & Goldberg, M. (2009). Effect of retirement on sleep disturbances: the GAZEL prospective cohort study. <i>Sleep</i> , 32(11), 1459-1466.	Dillon, H. R., Lichstein, K. L., Dautovich, N. D., Taylor, D. J., Riedel, B. W., & Bush, A. J. (2014). Variability in self-reported normal sleep across the adult age span. <i>Journals of Gerontology Series B: Psychological Sciences and Social Sciences</i> , 70(1), 46-56.

*Meta – analysis

APPENDIX B**www.sleeplessinphilly.com Complete Questionnaire****Screening Form****Directions for filling out the sleep and health information questionnaire:**

1. Please complete the entire form. Do not leave any question unanswered. If you are unsure about an item, please make a comment in the next "Comments" section of the form, so that we can obtain the most accurate data possible.
2. A few of the questions on the form may be considered by some as fairly personal. Please answer as truthfully as you can. No one but the staff of the University of Pennsylvania Behavioral Sleep Medicine Research Laboratory will have access to the information you provide and in most instances your personal data will be identified only by your social Security number or by a University of Pennsylvania Behavioral Sleep Medicine Research Laboratory subject number.
3. Once you begin the survey, you may take as long as you want, but you must complete the whole survey (without back paging) or your data will be lost.
4. When you are finished, don't forget to press the "submit form" button on the bottom of the questionnaire.
5. **Bold** fields are required.

SECTION 1 of 6: GENERAL INFORMATION

1. First Name:

2. Middle Initial:

3. Last Name:

4. Primary phone: _____
(Please indicate Home, Work, or Cell)

5. Secondary phone:
(Please indicate Home, Work, or Cell)

6. Tertiary Phone:
(Please indicate Home, Work, or Cell)

7. Best Time to Call:

8. E-mail Address:

9. Address 1:

10. Address 2:

11. City:**12. State:** _____**13. Zip/Postal code:****14. What is the best way to reach you? (Circle one):**

- E-mail
- Home Phone
- Work Phone
- Cell Phone
- Mail

15. Where did you hear about us?: _____**16. Sex (Circle one):** Female/Male**17. Date of Birth (MM/DD/YYYY):****18. Height:** _____ Feet _____ Inches**19. Weight:** _____ lbs.**20. What is the ethnicity with which you MOST CLOSELY identify? (Circle one):**

- Hispanic or Latino
- Not Hispanic or Latino

21. With which race do you MOST CLOSELY identify? (Circle one):

- American Indian or Alaska Native
- Asian
- Black or African-American
- Native Hawaiian or Other Pacific Islander
- White

22. Handedness (Circle one): Right/Left/Both**23. Education (Circle one):**

- No High School
- Some High School
- High School/GED
- Some College
- Associate's Degree

- Bachelor's Degree
- Master's Degree
- Doctoral Degree

24. Occupation (Circle one):

- Professional/Technical/Managerial
- Clerical/Sales
- Service
- Processing
- Structural Work
- Miscellaneous
- Unemployed
- Disabled
- Other

25. Typical Hours at Work (Please indicate hour and minute; Please circle AM or PM)

Start Time: : AM/PM

Stop Time: : AM/PM

26. Do you ever work rotating or split shifts (circle one)? Yes/No

27. Have you ever participated in a sleep study before (circle one)? Yes/No

28. Would you be willing to spend at least two overnights at the sleep research laboratory (circle one)? Yes/No

SECTION 2 OF 6: BASIC SLEEP/INSOMNIA INFORMATION

For the next four items, please indicate hours:minutes; Please circle AM or PM.

29. I typically go to bed at: _____ : _____ AM/PM on weekdays

30. I typically get out of bed to start the day at: _____ : _____ AM/PM on weekdays

31. I typically go to bed at: _____ : _____ AM/PM on weekends

32. I typically get out of bed to start the day at: _____ : _____ AM/PM on weekends

33. On a typical night, how many minutes does it take you to fall asleep?

34. How many nights a week (7 days) does it take you more than 30 minutes to fall asleep? _____

35. Do you consider this a problem (circle one)? Yes/No

36. How old were you when this first became a problem? _____ years old
37. On a typical night, how many times do you awaken in the middle of the night but fall back asleep?
38. How many nights a week do you awaken this often? _____
39. Do you consider this a problem (circle one)? Yes/No
40. How old were you when this first became a problem? _____ years old
41. On a typical night, how long are you awake altogether during awakenings?
minutes
42. How many nights a week are you awake for 30 minutes or more?
43. Do you consider this a problem (circle one)? Yes/No
44. How old were you when this first became a problem? _____ years old
45. Do you sometimes wake up before you intend to in the morning (circle one)?
Yes/No
46. Typically, how many minutes before?
47. How many mornings a week do you wake up 30 minutes early or more?
48. Do you consider this a problem (circle one)? Yes/No
49. How old were you when this first became a problem? _____ years old
50. On a typical night, how many hours of sleep do you get?
51. How many nights a week do you get this much sleep?
52. Do you consider this a problem (circle one)? Yes/No
53. How old were you when this first became a problem? _____ years old
For the following questions, please circle yes or no.
54. If you can go to bed and get out of bed any time you choose, can you sleep as much as you need to? Yes/No
55. Do you have difficulty with attention or concentration during the day? Yes/No

56. Are you irritable during the day? Yes/No

57. Do you tend to have an upset stomach or other GI problems during the day?
Yes/No

58. Do you feel rested when you wake up? Yes/No

59. Do you feel your daytime function is impaired due to trouble sleeping? Yes/No

60. Do you feel fatigued during the day? Yes/No

61. Do you feel sleepy during the day? Yes/No

62. Do you nap during the day? Yes/No

63. Do you consider trouble sleeping to be a problem for you? Yes/No

64. Did the onset of your trouble sleeping follow a specific event? Yes/No

65. Have you previously been treated for sleep problems? Yes/No

SECTION 3 OF 6: SLEEP RELATED QUESTIONS

66. Have you been told that you snore loudly (circle one)? Yes/No

67. How many nights per week do you snore loudly? _____

68. Have you ever been told that you momentarily stop breathing or that you seem to hold your breath while sleeping (circle one)? Yes/No

69. Do you sometimes awaken at night gasping for breath or with difficulty breathing (circle one)? Yes/No

70. Do you often wake up with a headache (circle one)? Yes/No

71. Do you often feel that you are extremely sleepy during the day such that you find yourself falling asleep, or fighting the urge to fall asleep, at inappropriate times and/or places (circle one)? Yes/No

72. Do you experience sudden attack of sleep during the day (one second you're awake, another you are asleep) (circle one)? Yes/No

73. Do you sometimes experience muscular weakness if you have a strong emotion (circle one)? Yes/No

74. Do you sometimes awaken from nighttime sleep with terribly frightening dreams (nightmares) (circle one)? Yes/No

75. Do you notice an unpleasant feeling (creepy crawly) or sensation in your legs as bedtime approaches (circle one)? Yes/No

SECTION 4 OF 6: GENERAL HEALTH QUESTIONS

76. Are you currently taking any prescription medications, over-the-counter medications, vitamins, or herbal supplements (circle one)? Yes/No

77. Please list any medications to which you have had an allergic reaction and describe the reaction you had:

78. Please describe any other allergies:

Do you currently or have you ever had any of the following medical conditions? Please circle one.

79. Epilepsy/ serious head injury (blacked out): Current/No/Past

80. Cancer: Current/No/Past

81. Heart disease (angina, pacemaker, CABG): Current/No/Past

82. Kidney disease: Current/No/Past

83. Liver disease/Hepatitis: Current/No/Past

84. Diabetes: Current/No/Past

85. Thyroid disorder: Current/No/Past

86. Nocturia (frequent awake to pee/urinate) or prostate disorder: Current/No/Past

87. Do you have fibromyalgia? Yes/No

88. Other: Current/No/Past

89. If yes, please describe:

For Women

90. Are you currently pregnant or breast feeding? ☐ Yes ☐ No
91. Do you currently have plans to become pregnant soon? ☐ Yes ☐ No
92. Have you reached menopause? ☐ Yes ☐ No
93. If yes, has it been more than two years since you have had symptoms of menopause?
☐ Yes ☐ No
94. Do you regularly use birth control? ☐ Yes ☐ No

Have you ever been diagnosed with the following psychiatric conditions? Please circle one.

95. Depression: Current/No/Past

96. Anxiety Disorder: Current/No/Past

97. Obsessive Compulsive Disorder: Current/No/Past

98. Schizophrenia: Current/No/Past

99. Bipolar Disorder or Manic Depression: Current/No/Past

100. Severe memory problems: Current/No/Past

101. Have you EVER taken any psychiatric medications? Yes/No

102. Have you ever been hospitalized for psychiatric reasons? Current/No/Past

103. Has anyone in your family ever suffered from a mental illness such as bipolar disorder or schizophrenia? Yes/No

104. How often did you have a drink containing alcohol in the past year?

- Never 0
- Monthly or less 1
- Two to four times a month 2

- Two to three times a week 3
- Four or more times a week 4

105. How many drinks do you have on a typical day when you were drinking in the past year?

- 1 or 2 0
- 3 or 4 1
- 5 or 6 2
- 7 to 9 3
- 10 or more 4

106. How often did you have six or more drinks in one occasion in the past year?

- Never
- Less than monthly
- Monthly
- Weekly
- Daily or almost daily

SECTION 5 OF 6: CHRONIC PAIN QUESTION

107. Do you suffer from chronic pain (circle one)? Yes/No

SECTION 6 OF 6: TREATMENT FOR INSOMNIA/SLEEPLESSNESS

108. Have you ever used a sleeping pill (hypnotic) (circle one)? Yes/No

PERMISSION TO REATIN INFORMATION FOR FUTURE USE

Please check the appropriate box after reading the statement below

I give my permission to the UPenn Behavioral Sleep Medicine Research Laboratory to retain the information I have provided so that I might be contacted about current and/or future opportunities to volunteer for research. I am aware that I can withdraw this consent at any time in the future and that

1. This will result in the removal of any and all information obtained about me from the laboratory's records (the hard copy binder, the online SIP form folder, the database, which contains subject contact information and primary designation Information. And

This will not affect, in any way, my care within the Univ. of Pennsylvania Health Care System. ___ Yes, I give my permission ___ No, I DO NOT give my permission